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ACTA RADIOLOGICA

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ACTA RADIOLOGICA
EDITA IUSSU SOCIETATUM RADIOLOGICARUM
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VOL. 45 MARCH 1956 FASC. 3

THE IMPORTANCE OF THE THORACIC DUCT IN THE SPREAD OF MALIGNANT DISEASE

by

Alejandro Celis, José Kuthy and *Ermilo del Castillo*

Our purpose in initiating this investigation was to find a means of rendering the lymphatics of the thorax opaque; for this purpose an anatomic and radiologic study was carried out in fifty human cadavers. After the preliminary report had been published (3) we completed the study in a further twenty-seven cases and arrived at the conclusion that in most cases the thoracic duct is a single trunk of variable diameter, tortuous, and constricted at intervals; it ascends through the posterior mediastinum, either in front or to the left side of the vertebral bodies, and rises in an arch a few centimetres above the left subclavian vein in which it terminates. As to the anatomic and radiologic variations, we noted a double trunk, a duct ending in the right subclavian vein or in both the right and left subclavian veins, branches with the azygos vein, and communicating branches with lymph nodes.

BIERMAN and associates (1), in 1953, reported the radiologic examination of the thoracic duct in ten patients with leukemia; these investigators introduced a catheter through the terminal portion of the duct in the neck and rendered it opaque by the injection of a contrast medium. We believe that the technique we have used in our studies in the human cadaver and living dog, as well as the technique employed by BIERMAN and his associates, are not applicable as a routine clinical procedure.

Following our studies in the cadaver, we began to work on the thoracic duct in the living dog; for this purpose we used forty-seven animals in which we introduced a catheter through the terminal portion of the duct in the neck. In some cases the presence of valves pre-

Submitted for publication 25 May 1955.

From the Tuberculosis Service and the Department of Experimental Surgery, General Hospital, Mexico, D. F.

a b

Fig. 1. Thoracic duct in the dog, a) A. p. b) Lateral. A lymph node together with some branches is rendered opaque.

vented any advancement of the catheter, while in others it was possible to reach the cisterna chyli.

The radiologic study of the thoracic duct in the dog was conducted in the a. p. and lateral projections. In the former we found, in most cases, a single trunk without branches arising in the abdomen in a saccular formation (cisterna chyli), ascending to the thorax to the left side of the vertebral bodies, and terminating in the neck at the level of the catheter (Fig. 1). In the lateral projection the contrast-filled thoracic duct consists of two distinct parts — abdominal and thoracic. In the first part, the duct arises in a saccular formation situated in the upper third of the abdomen and just in front of the vertebral bodies; it ascends through the diaphragm where its diameter is always diminished; the thoracic portion consists of a single trunk, although frequently more than one trunk, ascending in front of the vertebral bodies and terminating in the neck at the level of the entrance of the catheter, is found.

The most frequent variations we have met are: the presence of abdominal branches, several branches along the main trunk, and the formation of a dilatation at the inferior portion of the duct in the thorax; this latter, we believe, is due to the presence of a valve which impedes the downward flow of the contrast medium. In several instances it was possible to render a number of communicating branches and several lymph nodes opaque (Figs. 1 and 2).

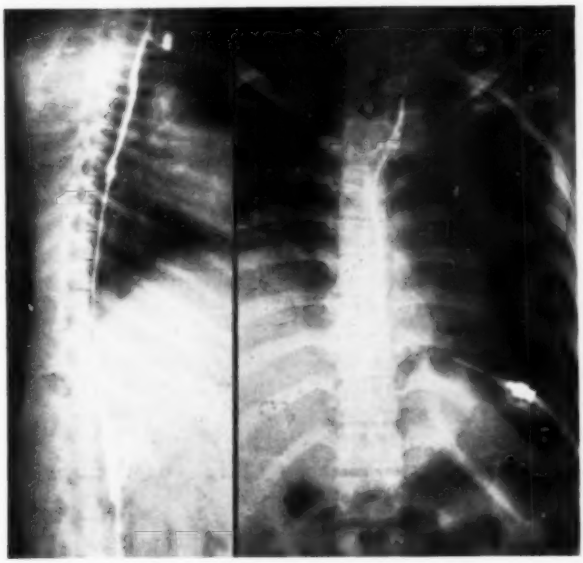

Fig. 2 Fig. 3

Fig. 2. Thoracic duct in the dog. The cisterna chyli is readily visible in the upper third of the abdomen. A lymph node has been rendered opaque in the upper part of the thorax.

Fig. 3. Thoracic duct in a case of uterine carcinoma. Supraclavicular lymph node metastases and malignant cells were found in the duct.

Based on the anatomic and radiologic studies of the normal thoracic duct, several pathologic conditions including pulmonary tuberculosis, cirrhosis of the liver, and secondary malignant tumours of the lung, were investigated; forty-four human cadavers were studied, in twenty-six of which cancer was the cause of death.

Our studies of the thoracic duct in cases of malignant disease are, with due regard to the following statistical data, of special interest: WILLIS (8) in 1,000 cases of metastases secondary to carcinoma found that 30 % were pulmonary; MINOR (6) reviewing the records of the Michigan Hospital between the years 1937 and 1942, recorded 5,727 cases of primary carcinoma of which 314 (5.7 %) showed pulmonary metastases; BRANDT and PEREZ TAMAYO (2) from the Department of Pathology, National University of Mexico, in 76 autopsy cases with carcinoma found that 20 (27.7 %) had metastases in the lung. Reviewing the autopsy records of our hospital, we were able to collect 86 cases with carcinoma of which 20 (23.2 %) showed secondary involvement of the

lung. Thus, for three or four patients dying of carcinoma, one will have metastases in the lungs.

There are two ways by which malignant new growths may reach the lungs: by the hematogenous and the lymphogenous routes, of which the former is more important and frequent.

The lymphatic spread of malignant new growths in the stomach, mammary gland, ovaries and adrenals, more frequently occurs through the posterior mediastinal lymph nodes in connection with the abdominal lymph nodes and thence to the hilar, bronchial, and blood vascular lymph nodes of the lung. The hematogenous route is by way of the venous circulation to the right heart chambers and thence to the lungs; through the hepatic veins and inferior vena porta; or by the lympho-hematogenous route through the thoracic duct, innominate vein, right heart chambers, and pulmonary circulation. We believe this latter route to be of great importance and that it explains a high percentage of metastases in the lung. It is true that epithelial type of new growths invade the lymphatic system, while those of the connective tissue are disseminated mainly through the hematogenous route although a great number invade the lymphatic system.

The thoracic duct carries most of the lymph of the body with the exception of the right side of the head and neck, right arm, and most of the structures contained in the thorax. If a malignant cell enters the lymphatic stream it will be conveyed by way of the thoracic duct to the venous circulation. It is also important to remember that the incidence of pulmonary metastases differs according to the site of the primary tumour. Thus, in the 76 cases reported by the Department of Pathology of the National Univ. of Mexico, 14 cases were suprathoracic and only one (7.1 %) had metastases in the lung, while the incidence of metastases rose to 32.2 per cent in those cases in which the primary tumour was situated below the diaphragm. In our series, 40 per cent of the cases with pulmonary metastases followed a primary growth situated below the diaphragm. The figures given by WILLIS (8) also stress the fact that with the exception of thoracic new growths and those of the thyroid gland, which spread by way of the venous circulation, there is a marked difference in the incidence of pulmonary metastases depending upon whether the primary tumour is situated above or below the diaphragm.

The third important fact to consider is the frequency with which the thoracic duct is invaded. WILLIS (8) in 81 autopsy cases found thoracic duct involvement in 2.8 per cent, the incidence being 6.1 per cent for a primary lesion situated below the diaphragm. WASHBURN (7) reported four cases of thoracic duct invasion in fifty autopsy records. YOUNG at the Congress on Pathology in Washington Sept. 1954 reported 86 cases in 40 per cent of which he found thoracic duct involvement; in 9 of these

86 cases the primary growth was situated above the thorax and in none of these was he able to find invasion of the thoracic duct; in 34 of his cases the primary tumour was either esophageal or bronchial with thoracic duct involvement in 21 (61.7 %). The explanation for this high incidence of thoracic duct involvement is not difficult — it occurs by direct extension; this is particularly the case with esophageal tumours since the duct is situated immediately to the left of the esophagus. Finally, in 43 cases in which the primary tumour was situated below the diaphragm, 14 (32.5 %) showed thoracic duct invasion.

A type of metastasis frequently useful in diagnosis is the secondary nodule in a left supraclavicular lymph node in association with carcinoma of one of the abdominal viscera, referred to as 'Virchow's node'; the thoracic duct is invaded and embolism or permeation to the node near the orifice of the duct in the innominate vein is the cause of this clinically prominent metastasis.

In the 86 cases reported by YOUNG, 38.3 per cent had a metastasis in the supraclavicular node, and all of them had thoracic duct invasion while in the cases with lymphoma, 92.3 % showed thoracic duct invasion. All these data suffice to show the importance of the thoracic duct in the spread of malignant disease.

Material and methods

Our purpose in continuing the current investigation was to study the thoracic duct in cases of malignant disease. For this purpose forty-four human cadavers were studied, in twenty-six of which cancer was the cause of death. The method we followed has been previously described (3). Soon after the thoracic duct had been rendered opaque and the roentgen investigations carried out, the cadaver was sent to the Department of Pathology where autopsy was performed.

Results

The cases may be divided as follows:

1. Cases in which the thoracic duct was normal. In none of these were there any malignant cells in the post-mortem examination of the duct. In only one instance in which the supraclavicular lymph node showed metastatic deposits and the radiologic picture of the duct had been normal, were there malignant cells in the inner walls of the duct (Fig. 3).

2. Cases in which there was obstruction of the duct. This obstruction may be situated in any portion of the duct and the radiologic appearances seem to indicate that this is an intrinsic type of obstruction; in most of these cases there were pulmonary metastases which can be explained

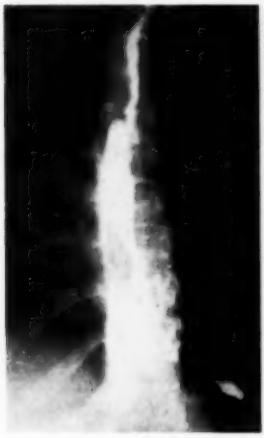

Fig. 4

Fig. 5

Fig. 4. Thoracic duct in a case of uterine carcinoma with supraclavicular lymph node metastases. Note dilatation in the middle part of the duct.

Fig. 5. Carcinoma of the larynx with supraclavicular lymph node metastases. Bifurcation of the duct and intercostal branches.

in that the embolus carrying the malignant cells passed through the duct into the lungs before the duct had been obstructed, or that the obstruction of the duct created new pathways ultimately terminating in a vein whereby the pulmonary metastases were formed.

3. The obstruction of the thoracic duct leads to the formation of new pathways or collaterals, or the visualization of those formerly not apparent; this collateral circulation may drain directly into the venous circulation or, after developing an intricate lymphatic network, finally drain into a vein. This collateral circulation may be demonstrated either by carefully dissecting the duct or filling it with contrast medium (Figs. 5 and 6).

Not in all the cases with obstruction of the duct could a collateral circulation be demonstrated. This may be due to the fact that not enough time passed between the obstruction and the death of the patient. The collateral circulations which are demonstrated in our roentgen studies of the duct involve mainly periaortic and periesophagic plexuses as well as intercostal branches.

We have been unable to find references in the literature to obstruction of the thoracic duct in autopsy cases of cancer with pulmonary metastases. The formation of a collateral circulation implies the possibility of the formation of metastases anomalous in their distribution.

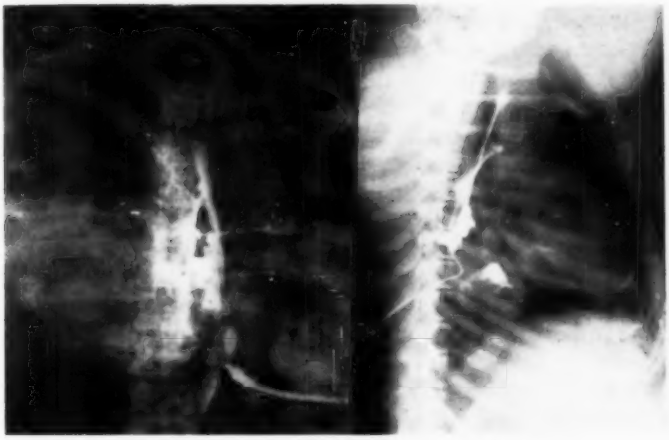

Fig. 6 Fig. 7

Fig. 6. Osteogenic sarcoma of the leg. The thoracic duct is obstructed. Formation of a collateral circulation (pulmonary metastases).

Fig. 7. Thoracic duct in the dog; ligated in the neck six weeks previously. Formation of a collateral circulation terminating in a vein.

The finding of obstruction of the duct in these cases introduces a series of problems such as the nutritional and hematologic alterations brought out by the obstruction. In order to attempt to elucidate these problems, we ligated the thoracic duct in a number of dogs.

The experimental ligature of the thoracic duct has been performed by several investigators. LEE (5) points out the fact that a collateral circulation develops between the seventh and fourteenth days after the duct has been occluded. CO TUI and associates (4) in their studies of the ligated duct, were able to demonstrate its importance in the return of proteins to the blood stream.

Our main purpose in ligating the duct in the dog was to demonstrate the radiologic alterations brought by its obstruction: the formation of collateral channels and their connections with the venous system. For this purpose we performed the experimental ligature of the duct in the neck in twenty-two dogs: these were observed for periods ranging from 15 days to 3 weeks, after which time the duct was approached through a left thoracotomy in order that it might be injected with contrast medium at the level of the diaphragm; in all cases we found the duct to be markedly dilated and in some cases a collateral circulation, mostly through the intercostal branches, was readily visible.

Of these 22 cases, 9 died after the ligation had been performed, some

showing post-operative complications such as infection; however, in two cases there were marked alterations in the lymphatic system, such as considerable dilatation of the duct and of the lymphatic channels in the diaphragm and omentum, which were filled with thick chyle; the small intestine was covered by a whitish dense band of chyle, there was evidence of a chylothorax, and a great quantity of chyle was present in the abdomen. Microscopically there was marked dilatation of the lymphatic capillaries on all of the abdominal viscera.

In the animals surviving ligation of the thoracic duct, a careful daily control of the lymphocytes of the blood and of the protein in the plasma was performed. Our findings were similar to those reported by other investigators in that there was a decrease in the number of the circulating lymphocytes and a hypoproteinemia, which returned to normal values in 4 to 5 days after the ligation. This we interpret as being due to the formation of a collateral circulation.

Roentgen studies in cases in which the duct was occluded by ligation of the terminal portion of the duct in the neck, showed the presence of anomalous lymphatic connections between the thoracic duct and the venous system, mostly in the upper portion of the thorax (Fig. 7).

Conclusions

From the anatomic and roentgen appearances of the thoracic duct in a number of human cadavers, as well as in cadavers with malignant new growths, and from the experimental ligature of the thoracic duct in the dog, certain conclusions appear justified. Apart from those classically accepted pathways for intrapulmonary neoplastic dissemination: hematogenous, lymphogenous and lymphohematogenous, the spread of malignant new growths through collateral channels between the lymphatic and the venous systems, after the thoracic duct has been occluded, is possible and this explains the formation of metastases which are anomalous in their distribution. Furthermore, it is generally accepted that following the involvement of the hilar ganglia the lung is also invaded by cancerous cells through retrograde lymphatic permeation; there is also the possibility that in some of these cases, the obstruction of the duct and the formation of collateral channels between it and the intrapulmonary lymphatic system may be the route followed by the cancerous cells in invading the lung parenchyma.

SUMMARY

A comparative study between the radiologic appearances of the thoracic duct in a number of human cadavers and in cases dying of carcinoma, in which the presence of obstruction of the thoracic duct and the formation of collateral channels which may

be the route for anomalous neoplastic spread, is described. The characteristics of the collateral circulation and the alterations brought by the obstruction of the thoracic duct are discussed.

ZUSAMMENFASSUNG

Eine komparative Studie über das röntgenologische Aussehen des Ductus thoracicus in einer Anzahl von Leichen und in Fällen, die einem Karzinom erlegen sind und bei denen die Anwesenheit einer Obstruktion des Ductus thoracicus und die Bildung von kollateralen Verbindungen den Weg für die abnorme Metastasierung des Neoplasmas gebildet haben mögen, wird beschrieben. Die Charakteristika der kollateralen Zirkulation und die Veränderungen, welche durch die Verengung des Ductus thoracicus entstehen, werden diskutiert.

RÉSUMÉ

Étude comparative entre les aspects radiologiques du canal thoracique sur un certain nombre de cadavres et sur des malades mort de cancer, chez lesquels sont décrites la présence d'obstruction du canal thoracique et la formation de canaux collatéraux qui pourraient être la voie suivie par des dissémination néoplasiques atypiques. Les auteurs examinent les caractéristiques de la circulation collatérale et les altérations apportées par l'obstruction du canal thoracique.

BIBLIOGRAPHY

1. Bierman, H. R., Byron, R. L., Kelly, H. K., Gilfillan, R. S., White, L. P., Freeman, N. E. & Petrakis, N. L.: The characteristics of thoracic duct lymph in man. J. Clin. Investigation 32 (1953), 637.
2. Brandt, H. & Perez, Tamayo, R.: Personal communication.
3. Celis, A. & Kuthy-Porter, J.: Lymphatics of the thorax. An anatomic and radiologic study. Preliminary report. Acta radiol. 38 (1952), 461.
4. Co Tui, Barcham I. S. & Shafiroff, B. G. P.: Ligation of the thoracic duct and the posthemorrhage plasma protein level. Surg., Gynec. & Obst. 79 (1944), 37.
5. Lee, F. C.: Cit. by Co Tui, Barcham & Shafiroff.
6. Minor, G. R.: A clinical and radiologic study of metastatic pulmonary neoplasms. J. Thoracic Surg. 20 (1950), 34.
7. Washburn, R. M.: Obstruction of the thoracic duct and chylothorax. Am. J. M. Sc. 196 (1938), 572.
8. Willis, R. A.: The spread of tumors in the human body. Butterworths & Co., London 1952.

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OSTEOCHONDRITIS ISCHIOPUBICA

by

Lars R. Holsti

Swellings in the region of the ischiopubic junction are fairly common in children and generally interpreted as normal. In the opinion of several authors, such changes are physiologic and ordinarily belong to normal ossification; they may occur, according to JUNGE and HEUCK (16), in every seventh healthy child. In various diseases of the hip-joint altered static conditions and greater stress upon the ischiopubic junction make the enlargements considerably more common (9, 11, 13, 16, 21, 22, 31, 35, 37), and according to the above mentioned authors they are found in two-thirds of all cases. Swellings are particularly common in congenital dislocation of the hip, Perthes' disease, and coxa vara, but are also seen in connection with other diseases of the hip-joint.

More than 30 years ago, however, attention was called to the fact that clinical symptoms may appear in association with swelling of the ischiopubic junction. The first cases were published by VAN NECK (25) in 1924: two girls of 8 and 11, respectively, had pain and swelling in the groin, restricted movement in one hip and a limp. The roentgenogram showed a round focus at the ischiopubic junction. Osteochondritis was demonstrated histologically in both cases. VAN NECK called the disease osteochondritis ischiopubica. In later years reports of similar cases have been scarce. A search of the literature at the disposal of the writer revealed a total of 95 cases (1, 4—11, 13, 14, 16—30, 33—38). Considerable confusion, however, appears to exist in the literature, since numerous cases published as osteochondritis ischiopubica are unmistakably of an inflammatory nature, and commonly osteomyelitis. In addition, there is some doubt as to what should be interpreted as normal and what as pathologic.

Clinical picture. A typical case of osteochondritis ischiopubica (o. i. p.) has a slow onset, without any particular cause or following slight trauma,

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with pain in the hip-joint and limping. Pain may be referred to the groin and the adductor muscles. It may be aggravated by over-exertion, walking, or standing. Limitation of motion of the hip, produced by spasm in the adductor muscles, is sometimes present. Local tenderness and swelling have also been found. According to McFADDEN (23), even in bilateral cases all the symptoms may be referred to one side. There is no fever in typical cases. The sedimentation rate is always normal. The symptoms are in no way characteristic, but almost invariably give rise to a suspicion of Perthes' disease, coxitis, etc. The true state of affairs is revealed by the roentgenographic examination.

Roentgenologic appearances. The first findings are decalcification, rarefaction, and cystic areas of absorption. Some weeks or months later, there is usually evidence of recalcification, increased density, and apparent bone regeneration (5). The course has been compared to that of aseptic necrosis (5, 24). A triangular or rounded swelling the size of a cherry or under is seen in the region of the ischiopubic junction and apparently projecting into the obturator foramen. The swelling has a cloudy or granular appearance, and translucencies are present; it is sharply circumscribed and the surrounding bone shows a normal structure without atrophy or periosteal deposits. According to DAVIDSON (7), the lesion arises from the ischium.

Etiology. The majority of authors have interpreted the disease as belonging to aseptic necrosis, *e. g.* HÄUPTLI (12). ELIASEN (10) was opposed to this view on the grounds that the ischiopubic synchondrosis is not an epiphysis with bone growth merely involving a process of ossification. Aseptic bone necrosis, however, is met with also in regions other than an epiphysis, for instance small bones in an apophysis and in some rare instances in the diaphysis as well. In addition, such a change has also been described in a symphysis (2).

Since cases of inflammation have also been published under the heading of this disease, it is only natural that an inflammatory etiology has been reported (8, 10, 23, 29), as well as complication by a secondary infection (1, 19, 26, 33).

PRATJE (31), by means of roentgenanatomic investigations, has gone a long way towards clarifying the *normal ossification* of the pubis and ischium. On an average the process is completed between the age of 9 and 11. PRATJE classified the process into 4 phases, of which Phase II, occurring from 4 to 6 years, is the most important and shows the synostosis formation proper. In Phase III, occurring between the ages of 7 to 10 ossification is already nearly complete. It is assumed that o. i. p. arises in the intermediate stage between Phases II and III, in which the ends of the bone make contact and osseous bridges begin to appear between them. If ossification is disturbed or delayed — mostly on account of

Fig. 1. Boy, aged $5^1/_2$. Swelling of right ischiopubic junction with irregular bone structure and cystic area.

changed static conditions — the presence of friction between the extremities of the bones has been assumed and considered to lead gradually to the formation of the protuberance (33). The onset of o. i. p. is most common between the age of 5 and 9 years, which just corresponds to the ossification process proper (PRATJE's II and III phases).

Own cases. The writer's material consists of a series of 15 cases seen in the Roentgen Department between 1946 and 1953. Of these, 13 were boys and 2 were girls, ranging in ages at the time of diagnosis from five and a half to twelve and a half years. None of these cases had fever and none had been treated in the hospital. One case exhibited no hip symptoms, but only vague discomfort in the lower abdomen; such cases have been reported previously (9, 12, 17, 24). The other cases are typical instances of o. i. p.

It would appear that various mechanical agents, such as trauma, overexertion of the hip, and changed static conditions, may in the first instance be cited as the causative factor. The writer found several such cases in his series, and two of these are reported in detail.

A boy, aged 8 years, had fallen one month previously while skiing down a hill, since when he had limped and complained of pain in the right hip. The roentgenograms showed normal hips, but both ischiopubic junctions and the ischium were enlarged, the right being the size of a cherry and with a cloudy structure in which translucencies were seen (Fig. 3). The symptoms rapidly subsided as soon as over-exertion was avoided.

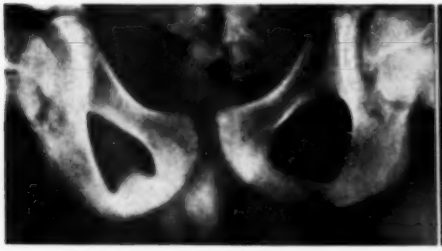

Fig. 2. Boy, aged 7. Right ischiopubic junction swollen with increased density of the bone and projecting into foramen obturatum.

Fig. 3. Boy, aged 8. Triangular osteoporotic protrusion into foramen obturatum from right ischiopubic junction. Demarcation lines distinct.

A girl of 8, having begun attending school, started limping and complaining of her right hip. The school was at a distance of 7 kilometres (about 4 miles), and the child walked there and back, a distance of 14 km daily. Examination revealed 1 cm shortening of the right lower limb, but nothing else of interest. The girl had never limped previously. Roentgenologic examination showed that both hip joints were normal. Enlargements were seen in both ischiopubic junctions. Avoidance of over-exertion was prescribed, and the girl was subsequently driven to the school and back; the symptoms receded swiftly. At a follow-up 5 months later she was completely free from symptoms and no longer limped. Ossification of the ischiopubic synchondrosis had progressed well.

Although several authors consider mechanical factors an important cause, the majority of cases reported in the literature began without manifest or demonstrable reason. There is, however, the possibility that in some of them at least slight and therefore neglected trauma had occurred. Moreover, in the majority of the cases reported, the patients were boys, who in general are probably more liable to slight mishaps.

The theory concerning the significance of mechanical factors is supported by the findings, already referred to, that swelling of the junction between the ischium and pubic are common *e. g.* in children with hip dislocations. Further support is lent by the fact that so-called march fractures are found in exactly the same region (15, 32).

The character of the disease is benign and the symptoms subside rapidly, often within a week or two, by avoiding exertion and without any special treatment. Roentgenologically the course of the disease is more prolonged, with a duration of several months, usually from 6 months to 1 year. The disease improves without leaving any traces. When ossification is completed, no deformation can be seen at the junction.

From the point of view of *differential diagnosis* in the roentgenologic sense one should primarily consider osteomyelitis and tuberculosis, although a tumour, osteitis fibrosa, and syphilitic osteochondrosis may be present in this region.

The *diagnosis* is based on the roentgenologic examination, but it cannot be determined solely by this means since clinical symptoms must be present as well. The greatest difficulty is in fact how to differentiate cases belonging to the group of physiologic ossification from those with disease processes. It is an indisputable fact that both these conditions exist, but in between them there is a group which it is not easy to place into the two foregoing. In several asymptomatic cases only bone thickening is seen roentgenologically, although there are also cases free from symptoms in which there is, in addition, irregularity in density of the bone. The writer had one case of his own, in which a boy, aged 7 $^1/_2$ years, had roentgenologically marked swelling and cloudy structure of both ischiopubic junctions, but clinically was entirely free from symptoms (Fig. 5). As CAFFEY (3) has remarked, it is noteworthy that normal or physiologic

Fig. 4

Fig. 5

Fig. 4. Boy, aged 6. Symmetric swelling of ischiopubic junctions, projecting triangularly into foramina obturata. The bone structure is partly decalcified, especially on the left side.
Fig. 5. Boy, aged $7^1/_2$. Bilateral swellings with irregular structure of ischiopubic junctions. Demarcation lines sharp.

irregular mineralization is found at many of the sites where osteochondrosis juvenilis or aseptic necrosis has been identified.

HEEREN (13) believed he could differentiate asymptomatic from pathologic cases in the roentgenogram. In cases which were attended with clinical symptoms he could in fact distinguish a sclerotic ring around the swelling, but this was absent in asymptomatic cases. However, JUNGE and HEUCK (16) at a later date found the calcification ring in asymptomatic cases as well, and believed that it was due to the tangential direction of the rays. At least for the time being, there are no reliable criteria from which it would be possible to differentiate roentgenologically between o. i. p. and cases of normal irregular mineralization. The differential diagnosis should consequently be founded on the clinical picture.

The significance of the o. i. p. as an independent disease is slight, but one should do well to bear it in mind for the purposes of differential diagnosis. If the hip-joint of a child complaining of symptoms in that region is found to be roentgenologically normal, the cause may possibly be discovered in the ischiopubic junction.

Acknowledgments

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SUMMARY

The author describes the clinical characteristics, and corresponding roentgen findings of osteochondritis ischiopubica, a benign disturbance of ossification in growing children, and reports 15 cases of his own. It is shown that it is not possible to differentiate between the condition and physiologically irregular mineralization upon the roentgenologic findings alone.

ZUSAMMENFASSUNG

Der Verfasser beschreibt die klinischen Merkmale der Krankheit sowie entsprechende Röntgenbefunde der Osteochondritis ischiopubica, einer gutartigen Ossifikationsstörung bei wachsenden Kindern und gibt einen Bericht über 15 eigene Fälle ab. Es wird gezeigt, dass es nicht möglich ist, ausschliesslich auf Grund der röntgenologischen Befunde die Differentialdiagnose zwischen diesem Zustand und physiologischer irregulärer Mineralisierung zu stellen.

RÉSUMÉ

L'auteur décrit les caractères cliniques et les signes radiologiques correspondants de l'ostéochondrite ischio-pubienne, trouble bénin de l'ossification chez les enfants en croissance, et en présente 15 cas personnels. Il montre que les signes radiologiques ne permettent pas, à eux seuls, de différencier cette affection de la minéralisation physiologiquement irrégulière.

REFERENCES

1. Asplund, G. A.: A few Cases of Ischio-Pubic Osteochondritis. Acta chir. Scandinav. 67 (1930), 1.
2. Burmann, M., Weinkle, I. N. and Langsam, M. J.: Adolescent osteochondritis of the symphysis pubis. With a consideration of the normal roentgenographic changes in the symphysis pubis. J. Bone & Joint Surg. 16 (1934), 649.
3. Caffey, J.: Pediatric X-Ray Diagnosis, First Edition. The Year Book Publishers. Chicago 1945.
4. Camerer, J. W.: Zur Differentialdiagnose der isolierten Erkrankungen des Schambeines. Kinderärztl. Prax. 13 (1942), 186.
5. Corper, F. J.: Osteochondritis Ischiopubica in Childhood. Am. J. Dis. Child. 56 (1938), 957.
6. Dainelli, M.: Su di un caso di osteochondrite della branca ischiopubica. Ref. in ZentOrg. ges. Chir. 58 (1932), 121.
7. Davidson, W.: Radiological Appearance and Clinical Significance of Osteochondritis Ischio-Pubica. Acta paediat. 11 (1930), 233.
8. Durham, H. A.: Ischiopubic Osteochondritis. J. Bone & Joint Surg. 19 (1937), 937.
9. Düben, W.: Umbauzone an der kindlichen Sitz-Schambeinverbindung und ihre differentialdiagnostische Bedeutung. Chirurg 21 (1950), 148.
10. Eliasen, P. N. B.: Nogle bemaerkninger om den saakaldte osteochondritis ischiopubica. (Danish.) Nord. med. 11 (1941), 2312.
11. Fairbank, H. A. T.: Ischiopubic osteochondritis. Brit. M. J. 1938, p. 148.
12. Häuptli, O.: Die aseptischen Chondro-Osteonekrosen. W. de Gruyter & Co., Berlin W 35, 1954.
13. Heeren, J.: Über röntgenologisch nachweisbare Veränderungen der Scham-Sitzbeinepiphyse und ihre klinische Bedeutung. Röntgenpraxis 5 (1933), 12.
14. Hirsch, A.: Über Osteochondritis ischiopubica. Kinderärztl. Prax. 4 (1933), 458.
15. Jones, D. B.: March Fracture of the Inferior Pubic Ramus: A Report of Three Cases. Radiology 41 (1943), 586.
16. Junge, H., and Heuck, F.: Die Osteochondropathia ischiopubica. Gleichzeitig ein Beitrag zur normalen Entwicklung der Scham-Sitzbeingrenze im Wachstumsalter. Fortschr. Röntgenstr. 78 (1953), 656.

17. Koch, Fr. and Wagner, H.: Die Osteochondritis ischiopubica, eine wenig bekannte Lokalisation der aseptischen Knochennekrosen. Monatschr. f. Kinderheilk. 100 (1952), 323.
18. Kohler, M.: Osteochondropathia ischiopubica. Kinderärztl. Prax. 22 (1954), 5.
19. Kroh: Die Osteochondritis ischiopubica. Zbl. Chir. 61 (1934), 2392.
20. Ledoux-Lebard, G.-R., Hecker, J.-P., Bonan, J. and Beavy, L.: L'ostéochondrite ischio-pubienne ou maladie de van Neck-Odelberg. J. de radiol. et d'électrol. 35 (1954), 419.
21. Longhi, L.: Sulla osteochondrite ischio-pubica. Ref. in ZentOrg. ges. Chir. 107 (1942—43), 383.
22. Manfredi, M.: Considerazioni su alcuni casi di osteochondrite ischiopubica. Ref. in ZentOrg. ges. Chir. 65 (1933—34), 632.
23. McFadden, G. D. F.: Ischio-Pubic Osteochondritis: with Report of a Case. Brit. M. J. 1938, p. 1309.
24. Meissner, K.: Osteochondritis ischiopubica. Tuberkulosearzt 5 (1951), 390.
25. van Neck, M.: Ostéochondrite du pubis. Ref. in ZentOrg. ges. Chir. 29 (1925), 469.
26. Nitter, L.: Osteochondritis ischiopubica. (Norwegian.) Nord. med. 37 (1948), 184.
27. Nordentoft, J.: Quoted by Eliasen.
28. Parisel, F.: L'ostéochondrite ischio-pubienne. Bruxelles-méd. 35 (1955), 438.
29. Petersen, K. E.: Osteochondritis ischiopubica. (Danish.) Nord. med. 13 (1942), 17.
30. Pollak, R.: Osteochondritis ischiopubica. Wien. med. Wchnschr. 84 (1934), 777.
31. Pratje, A.: Über die Ossifikation des Beckens. Verhandl. d. anat. Gesellsch., Erg.-H. zum Anat. Anz. 78 (1934), 53.
32. Schröder, W.: Die Ermüdungsfraktur an der Synostosis ischiopubica (schleichende Schambeinfraktur). Arch. f. orthop. u. Unfall-Chir. 42 (1943), 413.
33. Torgersen, J.: Osteochondritis ischiopubica. (Norwegian.) Norsk. mag. f. laegevidensk. 97 (1936), 951.
34. Valtancoli, G.: Osteochondrite ischio-pubica. Ref. in ZentOrg. ges. Chir. 32 (1925), 122.
35. Wilken, W.: Über die Osteochondritis ischio-pubica. Ztschr. f. Kinderh. 61 (1940), 127.
36. Wright, A. D.: Generalized Osteo-chondritis. Proc. Roy. Soc. Med. 24 (1931), 283.
37. Wülfing, M.: Über Osteochondritis ischio-pubica. Deutsche Ztschr. f. Chir. 199 (1926), 413.
38. Zeitlin, A. A.: Osteochondrosis-osteochondritis ischio-pubica. Radiology 27 (1936), 722.

FROM THE ROENTGEN DEPARTMENT (DIRECTOR: J. FRIMANN-DAHL), ULLEVÅL SYKEHUS, OSLO, NORWAY

ANGIOCARDIOGRAPHY IN INTRATHORACIC TUMOURS WITH PARTICULAR REFERENCE TO THE QUESTION OF OPERABILITY

by

Per Amundsen and *Edvin Sörensen*

Many authors judge angiocardiography to be of great value in the diagnosis and prognosis of primary lung tumours. DOTTER, STEINBERG and HOLMAN (1950), BATTEZZATI, SOANE and TAGLIAFERRO (1950), KEIL, VOELKER and SCHISSEL (1950) suggest that angiocardiography should be part of the routine examination in these cases. Others, *e. g.* WIKLUND (1951), hold that angiocardiography alone rarely yields information of value, especially concerning the question of operability.

Most of the authors up to date have used the technique introduced by ROBB and STEINBERG (1939, 1940), and have employed either no special equipment for film changing and injection of contrast medium or only a somewhat primitive method. However, during the last few years the technique in performing angiocardiography has improved considerably and is still improving. We feel that if this examination is used it must be performed in a way that will give all the information possible; the best technique available at the present time must be employed.

Material

We have reviewed the angiocardiograms performed in our department on patients suspected of having intrathoracic tumours. All those examinations which we felt were technically inefficient were discarded. We made this selection without knowing anything about the history, diagnosis, operability, etc. of the cases, and out of roughly 180 examina-

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tions we finally selected only 41 that we considered of sufficiently good technical quality to meet the present day criteria as a basis for diagnosis. All the discarded cases date back to the period 1948—1953, when we began our present technique.

Technique

The cases reviewed in the present series were all examined by some type of film changer, a few by manually driven home-made equipment, but most by the Schönander film changer which is in use at the present time.

As stressed by JÖNSSON et coll. (1949) it is necessary to ensure that the contrast medium passes through the vessels in a mass of high radio-opacity. The object is to visualise all the intrathoracic vessels from the innominate veins onwards, and we therefore inject the contrast medium through a polythylene catheter. This is introduced percutaneously into the basilic vein in the antecubital fossa according to the technique introduced by SELDINGER (1953). The tip of the catheter is generally placed in the subclavian vein just within the thoracic wall.

The injection is performed with an automatic pressure syringe (GIDLUND) at about 5 kg/m² which will generally allow the injection of 60 to 70 ml of medium within a little over 2 seconds.

Our present equipment permits exposure in only one plane and we therefore make two injections, using just under 1 ml Nycotrast 70 % per kg body weight. We allow about 15 to 20 minutes interval between the two injections so that no summation of the toxic effect of the contrast medium occurs (OLLE OLSSON 1950).

Before, during, and after, the injection a continuous recording is made including an electrocardiogram, together with a blood pressure tracing obtained through a polythylene catheter in the femoral artery; the injection and exposures are recorded upon the latter.

The injection is performed under a general anaesthetic. There are several reasons for this. The patient is spared the terrifying sensation following the injection, the anaesthetist is able temporarily to stop the breathing when required during the examination and, lastly but not least, there is full control of respiration should complications arise.

Complications

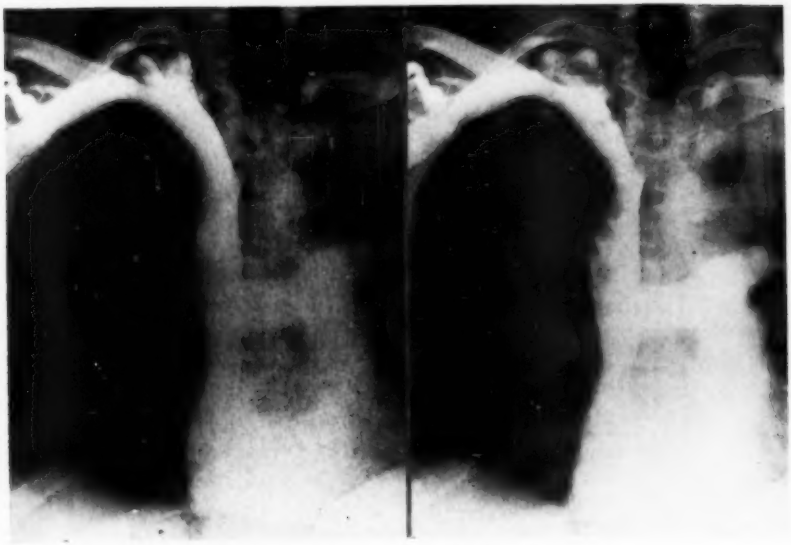
DOTTER and JACKSON (1950) state that in 6,824 angiocardiographic studies collected from 182 radiologic centres 26 deaths were reported. However, no death occurred in any patient with a healthy heart in the absence of renal disease. Practically all patients referred to angiocardiography because of suspected intrathoracic tumour will belong to this

group, and consequently one might estimate the risk to be negligible. Among our patients we twice observed ventricular arrest. In both, this lasted only for a short time and heart action restarted spontaneously. If a continuous electrocardiogram and blood pressure tracing had not been available we would certainly have failed to diagnose this asystole. We learned from this that even in this group of patients it is advisable to be prepared to meet the emergency of cardiac arrest. The treatment is artificial respiration with 100 % oxygen, thoracotomy, and direct cardiac massage. The result depends on the time elapsing between the onset of cardiac arrest and the beginning of effective heart massage. This period must not exceed 3 to 5 minutes, consequently an immediate and certain diagnosis is essential. It is difficult, however, to diagnose sudden cardiac arrest quickly and with certainty by simple clinical means (GYLLENSWÄRD and HOLMDAHL 1955) and that is why we suggest that a continuous electrocardiographic and blood pressure record is necessary during and after the injection. Not only will this give the diagnosis but will also serve as a control of the effectiveness of the treatment adopted. Where such a complication arises immediate planned action must be undertaken without discussion or any waste of time. Everything is therefore arranged beforehand, so that the anaesthetist looks after the respiratory side and the radiologist making the examination does the thoracotomy. Instruments for this purpose are always sterilized and ready.

Interpretation of the examination

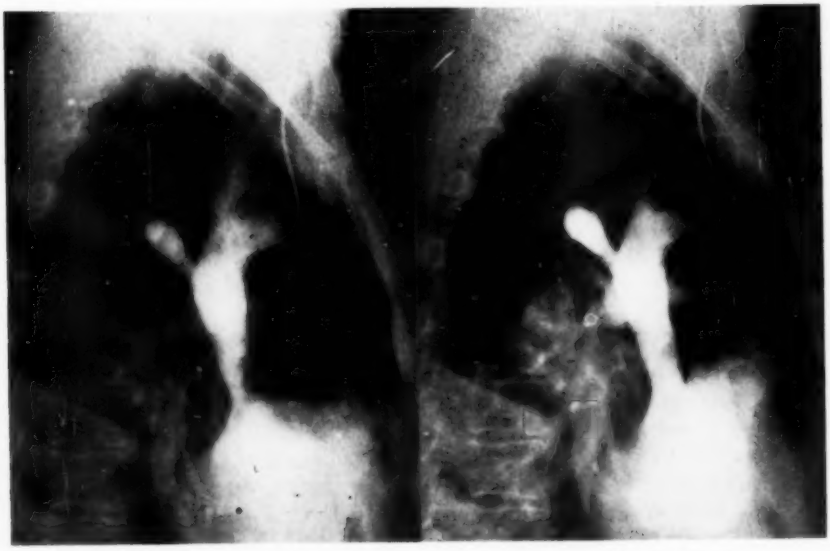
A diagnosis of filling defect or constriction of the great veins may be made erroneously from one single film, due to the failure of homogeneous mixing of the blood and the opaque medium on one side of the vein (Fig. 1 b) or to a physiologic constriction of the vena cava (Fig. 2 a). Another film from the film series made in a suitable cardiac phase will prevent this error (Figs. 1 a and 2 b). That is why we feel that serial exposures together with the knowledge of which point in the cardiac cycle each represents, are necessary.

Measuring the diameter of the main branches of the pulmonary artery will give some information. In all our cases we have measured the diameter of the main branches, the ascending and descending branch on the right side, and the difference in level between the right and left main branches (h. d.) (Table 1). The number of examinations upon which to base definite conclusions is too small, and moreover we have no normal figures for comparison. We suggest, however, that one may use as normal a group of cases presenting peripherally situated tumours which do not affect the central vessels and compare these with a group presenting centrally situated tumours on the left side and with another similar



a b

Fig. 1. a. End of systole. Vena cava of normal calibre and smooth contour. b. Late diastole. Defects in lateral contour of the superior vena cava due to admixture of blood from left innominate vein, shown in (a).



a b

Fig. 2. a. 'Constriction' of the vena cava during auricular systole. b. End of ventricular systole. Confirming the physiologic nature of the 'constriction'.

group on the right side. As to the left main branch, it is obvious that in left-sided centrally situated tumours the average figure for the diameter is reduced when compared with that for the two other groups. In 22 peripherally or centrally placed right-sided tumours the lowest figure is 1.8 cm and we think that a diameter less than this indicates narrowing. The mean figure for the h. d. is greater in both groups with centrally placed tumours than in the groups with peripherally placed tumours. The reason for this is that a tumour on the right side may depress the right main branch and on the left side may elevate the left main branch. There are, however, cases in the two groups in which the h. d. is less than in the control group owing to a tumour on the right side pressing the right branch

Fig. 3. Concentric narrowing of left main branch of pulmonary artery (1.4 cm) and displacement by a centrally placed tumour (difference in level between left and right branches 0.9 cm). Technically inoperable — confirmed.

Table I

Average diameter of left and right main branches of pulmonary artery, and average difference in level between the upper borders of these branches given for each group. (Figures in parenthesis: highest and lowest diameters measured)

	Number of cases	Average right main branch diameter (cm)	Average left main branch diameter (cm)	Average difference in level between lt. and rt. main branches. (h. d.) (cm)
Peripherally situated tumours	10	2.58 Range (2.3—3.4)	2.24 Range (1.8—3.4)	1.65 Range (1.1—2.5)
Centrally situated tumours (Left side)	8	2.90 Range (2.4—3.7)	1.65 Range (1.3—2.8)	2.08 Range (0.9—3.4)
Centrally situated tumours (Right side)	12	2.73 Range (2.0—3.2)	2.56 Range (2.1—3.1)	2.02 Range (0.9—3.5)

Fig. 4. Narrowing of left main branch of pulmonary artery (1.5 cm); only the lower border of vessel affected. Superior contour smooth; displacement upwards (difference in level 3.4 cm). No sign of centrally placed tumour. No circulation in left lower lobe from pulmonary artery because of occlusion of descending branch (Type 1). Pneumonectomy proved possible by pericardiotomy.

upwards or on the left side depressing the left main branch. We consider an h. d. greater than 2.5 cm or less than 1.0 cm indicates a displacement of one of the main branches.

There is no significant difference in the figure for the diameter of the right main branch. This is because this branch is very seldom narrowed as far medially as the point where we usually measure it. We find it valueless to measure the lesser branches as these measurements give no real information.

If the narrowing is concentric and occurs in the main branch centrally, close to the bifurcation, we feel there is no practical importance in knowing whether there is actual infiltration or merely constriction. Fig. 3 shows an example. The left main branch measures 1.4 cm, consequently it is narrowed and concentrically so. The h. d. is 0.9 cm, indicating a displacement of the left branch by a centrally placed tumour. We suggest that this sign is indicative of inoperability. A subsequent exploratory thoracotomy confirmed this diagnosis.

However, one has to be careful when only one side of the vessel is affected, and if there is no sign of infiltration medially. In the case represented in Fig. 4, the left main branch is narrowed, its diameter being 1.5 cm. The upper border is, however, smooth and the h. d. is 3.4 cm, indicating upward displacement of the left main branch. We concluded that the tumour was situated inferior to the vessel and that there was no sign of growth medially. This led us to predict that it would be technically difficult to isolate the main branch but we did not think that there were any definite signs of inoperability. At operation it proved possible to remove the tumour by opening the pericardium.

The circulation through the small vessels and capillaries in the lungs may be studied. For this purpose it is essential to use large films and a sufficient number of exposures. We believe this phase of the examination to be of considerable value. Diminution of the blood circulation through a whole lung or part of it may be caused principally at three points and accordingly we distinguish three types:

Type 1. Occlusion of the artery. Fig. 4 affords an example. There is no contrast medium in the lower lobe, indicating an occlusion of the descending branch. The upper lobe is atelectatic but there is contrast circulation through the pulmonary artery branches and the contrast medium is easily recognisable even in the veins from the upper lobe in the last film of the series. Nevertheless, occlusion of a peripheral branch does not *per se* indicate that the case is inoperable. Fig. 5 shows no circulation throughout the entire lung. This indicates a centrally placed occlusion and

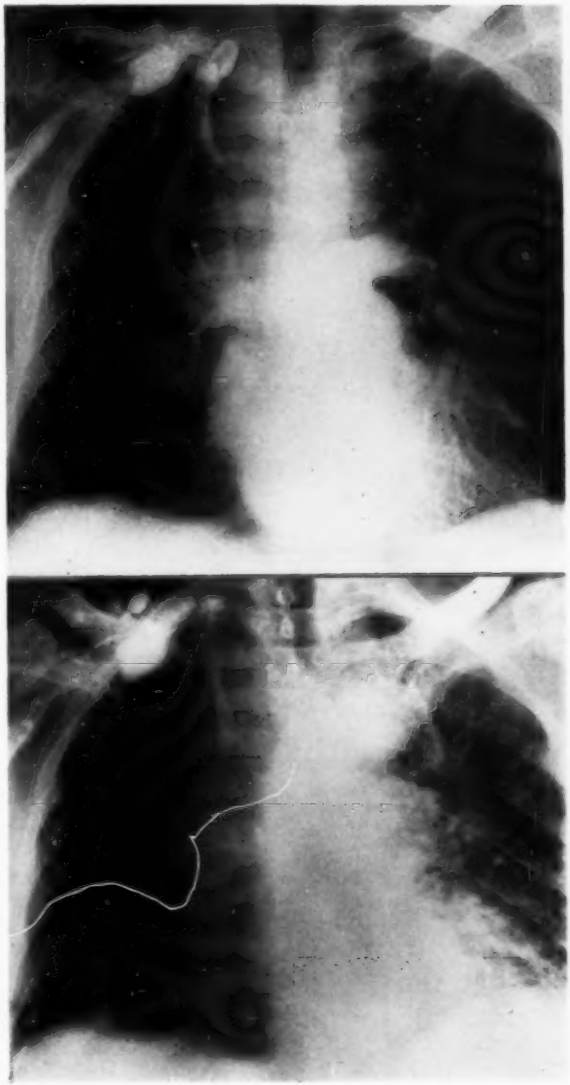


Fig. 5. *Top:* Pulmonary artery filled. *Bottom:* Pulmonary capillary and venous return. No circulation from pulmonary artery to right lung, indicating a central occlusion. In addition, superior vena cava involved. (Shown in the first films of series not presented here.) At autopsy 6 weeks later seen to be inoperable.

Fig. 6. Diminished circulation from pulmonary artery to left lung. Left main branch of pulmonary artery normal in calibre (2.2 cm); consequently not Type 1. *Diagnosis:* Bronchial adenoma in left main bronchus causing partial stenosis; an example of Type 2.

supports the view that the case is inoperable.

Type 2. Block in the peripheral circulation of the lung. The patient represented in Fig. 6 had an adenoma in the left main bronchus causing partial stenosis. It is obvious that the left main branch is of normal calibre (2.2 cm). The cause of the diminished blood supply passing through the left lung is consequently not an occlusion of the artery. Emphysema and probably other parenchymal diseases are capable of causing the same phenomenon. A reduced circulation of this type does not mean inoperability but rather supports the advisability of operating since it indicates reduced or abolished function of the lung.

Type 3. Block in the great lung veins. The diagnosis of this type must be made by excluding Types 1 and 2 as causes of the diminished circulation. Fig. 7 shows a normal diameter of the right main branch of the pulmonary artery as well as of the ascending and descending branches. Hence this is not a Type 1 case. The other examinations gave no indication of bronchial stenosis nor other pathologic condition in the lung. We feel that in view of these facts the diagnosis of an occlusion of the great veins of the lung is permissible. In this case there must be total occlusion of the lower veins, where no contrast medium can be detected, while the upper veins may be traced out in the later films in the series; they are only slightly constricted. An exploratory thoracotomy verified these findings. We feel that this type of diminished circulation is an important sign of inoperability. GIBBON et coll. (1948) state that the limits of operability may be extended in many instances by opening the pericardium and ligating the pulmonary veins flush with the left auricle.

However, in none of our cases showing occlusion of the great veins by angiocardiography did a radical operation prove possible.

Reduction of the circulation through a lung or part of it will cause diminished function and it is of great interest to correlate the results of bronchospirometry to the angiocardiographic findings.

The aorta and the brachiocephalic vessels seldom, in our experience, yield information of diagnostic value, as even large tumour masses may lie in contact with these vessels without producing sufficient distortion to show angiocardiographically. On the other hand they may serve as landmarks for tracing the pattern of the other vessels.

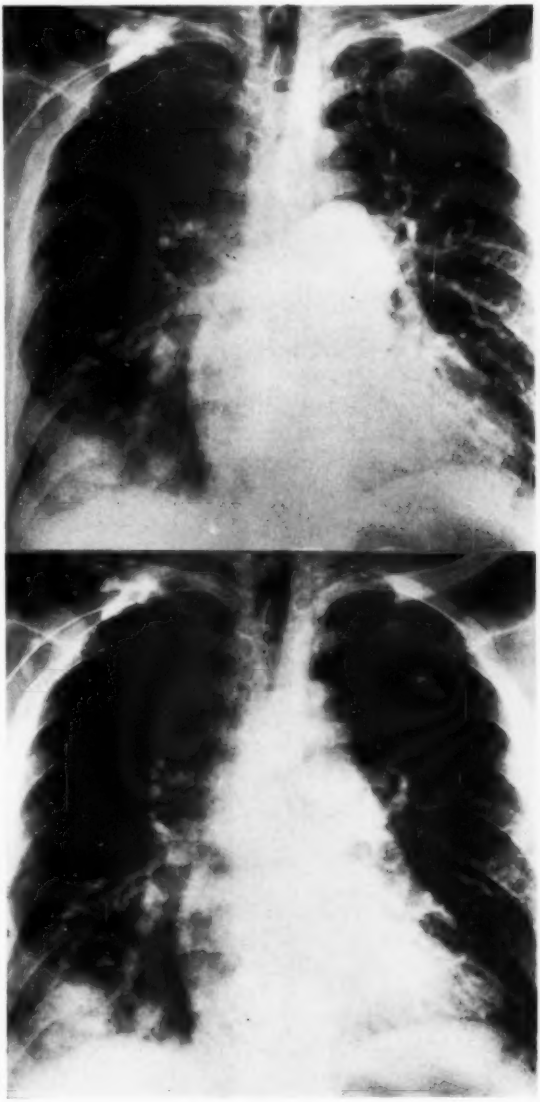


Fig. 7. An example of Type 3. Right lower pulmonary veins occluded and upper veins slightly constricted. On thoracotomy massive infiltration paratracheally and around the pulmonary veins, rendering case inoperable. Reduced circulation from pulmonary artery through right lung. Normal calibre of right main branch and of ascending and descending branches, thus not Type 1. No signs of bronchial stenosis or pathologic condition in lung, consequently not Type 2.

Indications for angiocardiography in intrathoracic tumours

1. *The nature of the condition.* In our experience angiocardiography is

of no help in deciding whether a pathologic process is due to neoplasm or not. One of our cases proved to be tuberculous but, on reviewing the examination we are unable to find anything that could distinguish this case from one of neoplasm. We would stress that the exact pathologic diagnosis must be made, if possible, with the help of other examinations such as planigraphy, bronchoscopy, biopsy, etc; only rarely does angiocardiography give a complete diagnosis. Even DOTTER and STEINBERG (1951) state that "it is frequently impossible to arrive with certainty at an opinion as to the neoplastic or infiltrative nature of infiltrative processes on the basis of angiocardiographically demonstrated vascular changes" although they add that an angiocardiographic diagnosis of lung cancer is possible in approximately 35 % of the cases studied.

2. *The question of operability.* With regard to lung tumour, the term 'inoperable' is neither exact nor well defined and interpretation of the term depends largely upon the surgeon. As a general principle we feel that one should be able to suggest inoperability from an examination in which the great vessels are either so extensively involved, or are involved locally in such a position as to render pneumonectomy or lobectomy impossible. Additionally, there may be signs of such extensive mediastinal involvement that the case will be beyond the scope of surgery. In certain groups of patients other examinations will suggest inoperability (metastases to other organs, bronchoscopic demonstration of infiltration of the carina, demonstration of massive growth by ordinary radiography or planigraphy, etc.). In such cases angiocardiography is contra-indicated since it can offer no additional information of practical value. Among those cases in which no other signs of inoperability can be detected, it is really worth while attempting to select the inoperable ones in order to save them from exploratory thoracotomy. This, although looked on as a safe procedure, nevertheless inconveniences the patient since it adds to his pain, discomfort, and suffering. Then again the avoidance of a thoracotomy means the saving of theatre work and nursing as well as beds.

3. *Preoperative information for the surgeon.* DOTTER and STEINBERG (1951) stress that angiocardiography gives the surgeon the anatomic extent of the lesion, information which must be of great help in planning and conducting the operation.

Our own material and conclusions

Signs of inoperability. According to DOTTER, STEINBERG and HOLMAN (1950) the criteria of inoperability are:

1. Complete or partial occlusion of the left main branch of the pul-

monary artery within 1.5 cm of its site of origin, or of the right main branch proximal to its point of bifurcation.

2. Partial or complete occlusion of the great mediastinal vein.

3. The diagnosis of mediastinal metastases by the demonstration of masses within the mediastinum through their external pressure effects on blood vessels.

4. The demonstration of pericardial invasion by tumour.

Twenty-three (23) of our cases proved to be inoperable and we picked out 17 of these, listed below, by angiocardiography. The numbers in parenthesis indicate the number of each group presenting other signs of inoperability.

Group 1.	Occlusion of great mediastinal veins.	2 (1)
» 2.	Complete or partial occlusion of right main pulmonary artery branch + Involvement of the superior vena cava.	3 (2)
» 3.	Complete or partial occlusion of left main pulmonary artery branch + Displacement indicating infiltration centrally.	4 (3)
» 4.	Occlusion of right ascending pulmonary artery branch. + Criteria of infiltration medially.	4 (2)
» 5.	Occlusion of pulmonary veins.	3 (0)
» 6.	Multiple tumours in both hila.	1 (1)
	Total	17 (9)

The small number in Group 1 is due to the fact that a tumour obliterating the vena cava will generally show other indisputable signs of inoperability and neither angiocardiography nor exploration is indicated. The same is true of Group 2. Our 3 cases in this group thus showed, in addition to occlusion of the right branch, distortion of the vena cava. However, in one of them no other sign of inoperability could be detected, angiocardiography being the only examination giving the diagnosis of inoperability prior to operation. Partial or total occlusion of the left branch (Group 3) is generally associated with a displacement of this artery, indicating a centrally placed tumour (Fig. 3). Obliteration of the subdivisions of the main branches (generally on the right side) is not a sign of inoperability unless, in addition, there is some other finding indicating involvement of the mediastinum (Group 4). The one error by DOTTER, STEINBERG and HOLMAN (1950) seems to be in just such a case, which did not, however, show any sign of mediastinal involvement. Nowadays one would not have regarded this case as inoperable.

Occlusion of the large vein (Group 5) is, in our opinion, important but it is, of course, essential that this diagnosis be indisputably accurate. In

all these cases angiocardiography is the only examination establishing the question of inoperability prior to thoracotomy. Group 6 should be omitted since the diagnosis is made by means of examinations other than angiocardiography.

Correlation between the angiocardiographic diagnosis and operative findings (Table II). Certain points are interesting. First, we find that in 8 out of 23 inoperable cases angiocardiography was the only examination establishing inoperability prior to exploration. In 7 of these cases, exploratory thoracotomy was performed; it confirmed the diagnosis, but showed that our surgeons dare not rely on angiocardiography alone as a criterion of inoperability. Secondly, we wish to emphasize that in no case did we make the mistake of stating that a tumour was inoperable only to find subsequently that radical extirpation might have been performed. We feel that this fact should justify more confidence in angiocardiographic findings where they suggest, without any reservation, that a case is inoperable. In those cases in which there are reservations, the patient should be given the benefit of the doubt and an exploratory thoracotomy should be performed — one of the two cases in this group proved to be operable.

Table II

Correlation between the angiocardiographic diagnosis and the operative findings in 37 cases of lung and mediastinal tumours

Inoperable	Angiocardiographic conclusions					
	Inoperable		Probably inoperable	Operable		
	No other signs	Other signs		No other signs	Other signs	
Exploratory thoracotomy ..	7	3	1	1	2	14
Not explored	1	6	0	0	2	9
Total	8	9	1	1	4	23
Operable	0	0	1	13	0	14
Total	17		2	18		37

Thirdly, 5 cases showing no angiocardiographic signs of inoperability subsequently proved to be inoperable. This fact has been repeatedly quoted. DOTTER and STEINBERG write: "The lack of vascular changes cannot be considered indicative of a favourable prognosis." However, 4 of these cases showed other signs pointing to inoperability. One must emphasize the fact that posteriorly situated tumours may not involve or influence the large vessels but will, not infrequently, invade the oesophagus and thus demonstrate their inoperability.

It seemed to us from a critical survey of 14 exploratory thoracotomy cases that 7 might have been saved operation from the angiocardiographic findings alone and that 5 more could have been added to this

number if we included other signs of inoperability along with the angiocardiographic ones. We feel, therefore, that in only 2 of the 14 cases was an exploratory operation essential.

Of 23 cases submitted for angiocardiography, 13 already had signs indicating inoperability and should not have been submitted to angiocardiography.

The conclusions we would draw from this material are in line with the experiences of DOTTER and STEINBERG:

1. The main indication for angiocardiography in tumours of the lung and mediastinum is to help to decide whether the growth is technically operable or not, and to give the surgeon information on the anatomic extent of the lesion. Angiocardiography should therefore be carried out on every case in which operation is intended. We think that many of these cases would thus, incidentally, be spared thoracotomy.

2. Those other cases showing unequivocal signs of inoperability should not even be submitted for angiocardiography.

3. Only occasionally, and in special cases, will angiocardiography give an indication of the nature of the lesion and we would emphasize that the diagnosis of neoplasm must be made on the basis of other examinations.

Angiocardiography is not an end in itself and does not render other examinations unnecessary. We would repeat that it is merely part of a complete routine, and should be the last examination performed prior to operation. Thus, reviewing our 37 cases and applying the principles enunciated above, we feel that altogether 13 angiocardiographies and 12 exploratory thoracotomies were unnecessary.

SUMMARY

The technique of, and indications for, angiocardiography in cases of suspected intrathoracic tumours are discussed in 41 angiocardiographically studied cases, 37 of which proved to have lung or mediastinal tumours. Seventeen (17) of these cases were judged by angiocardiography to be inoperable, an opinion verified in 12 by thoracotomy, the remaining 5 having no operation. Angiocardiography is of great value in cases of suspected intrathoracic tumour, especially where the question of operability arises, but probably of no real value in the actual diagnosis.

ZUSAMMENFASSUNG

Die Technik sowie die Indikation der Angiokardiographie in Fällen mit Verdacht auf intrathorakale Tumoren wird in 41 angiokardiographisch studierten Fällen diskutiert; von diesen hatten 37 Lungen- oder Mediastinaltumoren. Siebzehn dieser Fälle wurden mit der Angiographie als inoperabel beurteilt; diese Auffassung wurde in 12 Fällen durch eine Thorakotomie verifiiert, die übrigen 5 wurden nicht operiert. Die Angio-

kardiographie ist bei Verdacht auf intrathorakale Tumoren von grossem Wert, besonders in Fällen, wo es gilt, die Operabilität zu beurteilen. Für die aktuelle Diagnosestellung ist sie jedoch nicht von wirklicher Bedeutung.

RÉSUMÉ

Les auteurs discutent la technique et les indications de l'angiocardiographie dans les cas suspects de tumeur intrathoracique sur 41 cas étudies par angiocardiographie, dont 37 se sont révélés avoir des tumeurs pulmonaires ou médiastinales. Dix-sept (17) de ces cas ont été jugés inopérables d'après l'angiocardiographie, opinion confirmée dans 12 cas par la thoracotomie, les 5 autres n'ayant pas été opérés. L'angiocardiographie présente un grand intérêt dans les cas suspects de tumeur intrathoracique, particulièrement quand se pose la question de l'opérabilité, mais n'a probablement pas de réelle valeur pour le diagnostic lui-même.

LITERATURE

Battezzati, M., Soane, F. and Tagliaferro, A.: Die Angiocardiopneumografie zur Diagnose der Lungen- und Mediastinaltumoren. Schweiz. med. Wchnschr. 31 (1950), 799.

Dotter, C. T. and Jackson, F. C.: Death following angiocardiography. Radiology 54 (1950), 527.

Dotter, C. T. and Steinberg, I.: Angiocardiography. Annals of Roentgenology, Vol. XX. Paul B. Hoeber, Inc. Publishers. New York 1951.

Dotter, C. T., Steinberg, I. and Holman, C. W.: Lung cancer operability by angiocardiographic study of 53 consecutive proved cases of lung cancer. Am. J. Roentgenol. 64 (1950), 222.

Gibbon, J. H., Clerf, L. H., Herbut, P. A. and DeTuerk, J. J.: The diagnosis and operability of bronchiogenic carcinoma. J. Thoracic Surg. 17 (1948), 419.

Gyllenswärd, Å. and Holmdahl, M. H.: Hjärtstillestånd i samband med angiocardiografi. (Swedish.) Nord. Med. 53 (1955), 317.

Jönsson, G., Brodén, B. and Karnell, J.: Selective angiocardiography. Acta radiol. 32 (1949), 486.

Keil, P. G., Voelker, C. H. and Schissel, D. J.: Diagnostic value of pulmonary arteriography in bronchial carcinoma. Am. J. M. Sc. 219 (1950), 301.

Olsson, O.: Tolerance of cerebral blood vessels to contrast media of the Diodrast group in animal experiments and in man. Acta radiol. 34 (1950), 357.

Robb, G. P. and Steinberg, I.: Visualization of the chambers of the heart, the pulmonary circulation and the great blood vessels in man: a practical method. Am. J. Roentgenol. 41 (1939), 1.

Robb, G. P. and Steinberg, I.: Visualization of the chambers of the heart, the pulmonary circulation and the great blood vessels in man: summary of method and results. J. A. M. A. 114 (1940), 474.

Seldinger, S. I.: Catheter replacement of the needle in percutaneous arteriography. Acta radiol. 39 (1953), 368.

Wiklund, T.: Bronchiogenic carcinoma. Acta chir. Scandinav. Suppl. 162 (1951).

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ROENTGENOLOGIC SIGNS OF CHRONIC GASTRITIS

by

Kai Setälä and *Max Siurala*

The mucosal pattern technique has been widely used since its introduction in the roentgenologic examination of the stomach (1, 4, 5) in the diagnosis of various conditions. It has also been used for determining what is known as chronic gastritis (4, 11). The results, however, have not been encouraging (2, 3, 7, 12, 14, 16, 20, 21). The gastroscopic and histologic findings have varied considerably even in cases of roentgenologic 'tumour-forming' gastritis, which appears as gross changes in the mucosal relief often situated in the pyloric antrum and simulating gastric neoplasm. Gastroscopic and histologic examinations have in such cases revealed carcinoma, ulceration, gastritis, or even normal mucosa (6, 9, 14, 18, 21).

From the above facts, it is understandable that it has been rather difficult to obtain correlation in cases in which the pathoanatomic changes in the gastric mucosa have been moderate or slight. It has even been emphasised that the roentgenologic findings in the examination of the stomach reveal a patho-physiologic rather than a patho-anatomic condition (2, 12). This, of course, may be true in the case of the normal stomach and is believed to be in agreement with the teaching of FORSSELL (5). If, on the other hand, the function of the muscularis mucosae is the chief factor in determining the pattern of the gastric rugae, as FORSSELL has shown, it may be assumed that pathologic changes in the muscularis mucosae also cause changes in the rugal pattern. The former changes are known to be common even in more moderate forms of chronic gastritis (8, 14). These aspects, however, do not appear to have received sufficient attention in the literature. We would draw attention to certain

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aspects of many of the examinations reported. The roentgenologic findings have often been compared with the gastroscopic findings, without confirmation by biopsy. Gastroscopy, however, even if always performed by the same examiner, is a subjective method and the gastroscopic and histologic findings do not always agree (3, 21, 22). Moreover, the histologic diagnosis has been established from biopsy specimens removed during an operation or autopsy, in which post-mortem changes occur as greatly disturbing, sometimes even misleading, factors (15). A correct patho-anatomic examination is therefore essential for the evaluation of the relative importance of the roentgenology of gastritis. It is also evident that if different investigation methods are to be compared, the region examined must be clearly defined and be more or less the same in both examinations. In general, changes due to gastritis are limited to certain regions of the stomach; in another part of the same organ the changes may assume quite a different nature. In addition, it is essential that gastroscopic examinations be performed by the same person, so that there is some guarantee of the criteria being uniform. The requirements are much the same with roentgenologic examinations. It is equally essential that the microscopic evaluation of the biopsy specimens should always follow the same principles.

The present authors are of the opinion that if the above-mentioned sources of error can be excluded, some correlation will be found between the changes in the mucosal pattern and the patho-anatomic findings. We therefore tried to determine whether any common roentgenologic sign can be considered indicative of gastritis and in agreement with the gastroscopic and/or histologic appearance of the gastric mucosa, with particular reference to pathologic changes in the muscularis mucosae.

Material and methods

The material consisted of 401 cases gastroscopically grouped as follows (Table 1).

Table 1

Gastroscopic diagnosis

Mucosal condition	No. of Cases
Normal appearance	198
Atrophic gastritis	99
Superficial gastritis	47
Hypertrophic and mixed gastritis	57
Total	401

In all the 203 cases with gastritis, the mucosal changes were both histologically and gastroscopically moderate or slight in character. Excessive node formation or rigid and tortuous folds were not found in any case at gastroscopy.

In 302 cases of the material, biopsy specimens were taken from one or more regions of the gastric mucosa by means of our own instrument for gastrobiopsy (17). After the performance of gastroscopy the biopsy tube was introduced to the level corresponding to the upper region of Situation I and lower region of Situation II of SCHINDLER (13). Efforts were made to secure 3 specimens, one from the anterior wall, one from the posterior wall, and one from the greater curvature.

The series included 26 cases of peptic ulcer, and three of gastric carcinoma. In none of these was the lesion situated within or in the neighbourhood of the actual region.

The roentgenologic and gastroscopic examinations were all performed by the authors themselves, the operator performing the one type of examination being kept in ignorance of the results obtained by the other.

The *gastroscopic diagnosis* was based on the criteria presented by SCHINDLER (13). Accordingly, the following types of gastritis were used as basic groups: superficial, hypertrophic, and atrophic. Furthermore, cases with simultaneous atrophic and superficial changes were classed as superficial gastritis, with the exception of those with minute superficial changes — these being considered as atrophic. Cases with hypertrophic and mixed gastritis (*i. e.*, hypertrophic with superficial and/or atrophic changes) were placed in the same group. All the cases in which the gastroscopic alterations were only slight or strictly limited, were added to the group of normal cases. Gastroscopically only the aboral segment of the body — corresponding to SCHINDLER's (13) 'Situation I and II' — was taken into account.

The patho-anatomic diagnosis was based upon the following criteria:

Normal gastric mucosa. This group included all cases in which neither loss of the body glands, significant inflammatory cell infiltration, nor hyperplasia of the mucosa, could be observed. The histologic appearance of the normal gastric mucosa thus corresponded to that defined by SCHINDLER (14), and PALMER (10).

Atrophic mucosa. The group included cases in which loss of the body glands was so great that the zone of the main and the parietal cells was reduced to at least half the normal. In most cases there was a marked or moderate inflammatory cell infiltration of the entire gastric mucosa. In addition, intestinal metaplasia, or so called pseudopyloric metaplasia, occurred in about 60 % of the material.

Hyperplastic mucosa. This group included cases with somewhat thickened mucosa caused by hyperplasia of one or many of the mucosal elements. Excessive mucosal hyperplasia was not noted in any case. Clear inflammatory signs were present in about half the cases. The microscopic criteria of this group corresponded only partly to SCHINDLER's definition of chronic hypertrophic gastritis.

In all the groups mentioned there were cases with mucosal edema and inflammatory alterations beneath the surface epithelium. If these changes occurred in connection with the normal body gland layer, the case was considered as *superficial* gastritis, corresponding rather closely to a similar type described by SCHINDLER (14). Furthermore, cases with superficial inflammation in otherwise atrophic gastric mucosa are discussed as an atrophic-superficial group. The cases with these alterations in connection with hyperplastic mucosa formed a group which we call hyperplastic-superficial gastritis.

The *roentgenologic diagnosis* of gastritis was not made primarily because of the nature and the purpose of the present study. The roentgen examination of the stomach was always performed between 8 a. m. and 10 a. m., the patients having fasted for at least 12 hrs. No medication was permitted on the day of the examination or during the three previous days. General principles were followed in the roentgen examination which was performed with the patient in the erect, supine, and prone, as well as the Trendelenburg position. Since the correlation of certain areas of the gastric mucosa was in question, attention was paid especially to the area outside the costal margin. In the illustrative cases, the roentgenograms were selected by taking only these areas into account.

Obese or otherwise unsuitable subjects were not included in the series. The patients were instructed to swallow about 250 ml of a barium cream consisting of 50 g of barium sulphate per 200 ml of tap water, no emulsifying agent being used. The patients swallowed the cream in small draughts as instructed. The following signs were noted separately: form, size, tonus, and peristalsis of the stomach, as well as the amount of residual content, changes and/or appearance of the mucosal relief pattern, the presence or absence of the rugae, and regularity, continuity and thickening of the individual folds. The evaluation of some of these signs, especially when associated with turgor was naturally difficult in certain subjects. The χ^2-test was employed in the statistical calculations.

Results

The results for the entire series are given first and followed by illustrative cases.

When examining the occurrence of such roentgenologic signs as alterations in tonus and peristalsis of the stomach, as well as an increased residual content in *gastroscopically* different mucosal conditions, it became evident that there was only a slight and statistically insignificant difference between various types of gastritis and normal appearances. Peristalsis was increased relatively often (about 20 %) in cases with hypertrophic and mixed gastritis. Roentgenologic signs of an actual fluid content were observed in cases diagnosed as hypertrophic and mixed varieties relatively more frequently than in the others.

Table 2

Changes in the mucosal pattern in gastroscopically different conditions of the gastric mucosa

Mucosal condition	No. of Cases	Increased turgor	Appearance of folds		
			Coarseness	Irregularity	Thickening
Normal	198	16	9	22	40
Atrophic	99	6	4	8	26
Superficial	47	4	6	8	21
Hypertrophic and mixed	57	12	12	13	28
Total	401	38	31	51	115

Since no significant difference was noted between various roentgenologic signs in the preliminary examination, only actual roentgenologic alterations in the relief pattern were taken into consideration (Table 2). It became evident that the increased turgor of the folds, defined by palpation, occurred most frequently (about 20 %) in the cases with hypertrophic gastritis. It should be noted that this sign also occurred in the other groups of the material. Accordingly, it cannot be of very great diagnostic importance. Another relief syndrome (irregularity and coarseness of the folds) was relatively more frequent in the cases with gastritis in which the patho-anatomic finding corresponded to

superficial or to hypertrophic gastritis according to the above-mentioned criteria. The value of the result is diminished, however, by the fact that these changes also occurred remarkably often in the other groups. Likewise, the third sign (thickening of the mucosal folds) appeared relatively more frequently in cases with superficial or hypertrophic gastritis: in 50 % of the latter group as compared with 20 % of normal cases. (The difference was statistically significant.) In addition, thickening of the folds often occurred also in the other groups. As regards the occurrence of different signs in the relief pattern, there were no significant differences between the atrophic and normal groups.

Table 3 shows the occurrence of different roentgenologic signs in the relief pattern in various conditions of the gastric mucosa, verified histologically (302 cases).

Table 3

Changes in the mucosae pattern in histologically different conditions of the gastric mucosa

Mucosal condition	No. of Cases	Appearance of folds			
		Increased turgor	Coarseness	Irregularity	Thickening
Normal	109	5	4	11	23
Atrophy	92	9	4	9	25
Atrophic-superficial	24	2	4	6	15
Superficial	28	5	1	5	15
Hyperplastic	37	11	5	19	25
Hyperplastic-superficial	12	2	1	2	6
Total	302	34	19	52	109

Tonus and peristaltic disturbances as well as an increased fluid content are not shown in Table 3 since no noteworthy differences were present in various histologic subgroups. Table 3 reveals that increased turgor occurred relatively more often in the hyperplastic group (though even then only in 27 % of cases). Irregularity of the folds was observed relatively more frequently in the hyperplastic subgroup (about 50 %) than in the normal (about 9 %). (The difference was statistically significant.) The differences in coarseness were not so marked. Thickening of the folds was observed in 55 % of the cases with superficial inflammation (atrophic-superficial and hyperplastic-superficial groups, and superficial gastritis), in 61 % of the cases with hyperplasia of the gastric mucosa (hyperplastic and hyperplastic-superficial group) and only in 21 % of the normal group. (As compared with the normal group the difference was of statistic significance.) Only slight and insignificant differences were noted in the different relief changes between cases with atrophy and the normal group.

In accordance with the views presented in the introduction, special attention was drawn to the condition of the muscularis mucosae and its

correlation with the relief pattern. Only cases with perfectly preserved muscularis mucosae were taken into account. These totalled 123. The results are presented in Table 4. The muscularis mucosae was regarded as pathologic when it was definitely thickened, irregularly split up by inflammatory cell infiltration and a considerably increased amount of connective tissue, and when the boundary between the muscularis mucosae and the tubules was obscure and thick muscle and connective tissue bundles grew from the muscularis mucosae into the mucosa. The changes in the muscularis mucosae were divided into slight or moderate, and marked.

Table 4

Comparison of patho-anatomic changes in the muscularis mucosae with the relief findings

State of muscularis mucosae	No. of Cases	Normal relief	Increased turgor	Coarseness and/or irregularity	Thickening of folds
Normal	32	29	1	1	3
Moderate or slight changes	61	40	7	6	20
Marked changes	30	15	7	3	12
Total	123	84	15	10	35

It appears from Table 4 that the results are in fair agreement with those in Table 3. Changes in the mucosal pattern, especially thickening of the folds, are present only in a few cases with a normal muscularis mucosae and in half of the cases with severe alterations in the muscularis mucosae. (The difference was statistically significant.) Gross changes in the mucosal pattern were found in 2 cases with marked, in 2 cases with moderate or slight alterations in the muscularis mucosae, and in none with a normal muscularis mucosae.

The series included 20 cases with *marked changes* in the mucosal pattern. Gastroscopically the mucosa appeared normal only in 4 cases, atrophic gastritis was present in 2, and hypertrophic and/or superficial gastritis in 14 cases. Histologically, alterations due to gastritis were present in all of these. In 5 of the cases they were of the atrophic, and and in 15 of the hyperplastic and/or superficial type.

Illustrative cases

Two cases with normal roentgen appearances of the mucosal relief are reported. In Case 1 the gastroscopic and histologic findings were also normal. In the other, marked atrophic gastritis with signs of superficial inflammation were present in spite of the normal roentgen appearances.

Case 1 (1074/52). Female, aged 32, with pain in the lower abdomen of a few months' duration. Clinical examination: N. A. D. Histamine test-meal showed free hydro-

Fig. 1. Case 1. Normal mucosal pattern.

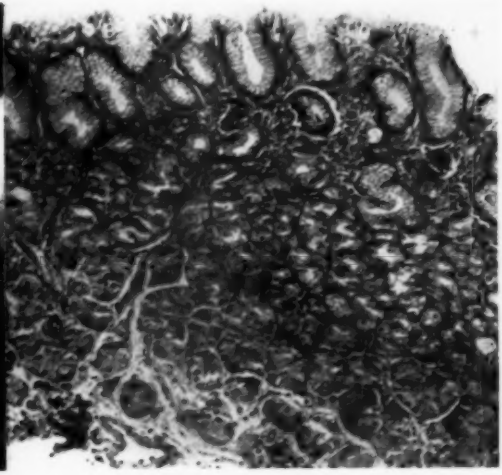

Fig. 2. Same case. Normal gastric mucosa.

Fig. 3. Case 2. Gastric rugae smooth and continuous.

Fig. 4. Same case. Slight alteration in surface epithelium. Round cell infiltration and edema. Thickening and irregularity of muscularis mucosae.

chloric acid. *Roentgen examination of stomach:* Normal folds within the correlation area. The aboral part of the body, where the folds were not visible, was outside the correlation area (Fig. 1). *Gastroscopy:* Normal mucosa. Histology: Normal gastric mucosa (Fig. 2).

Fig. 5. Case 3. Slight changes in the gastric mucosal pattern.

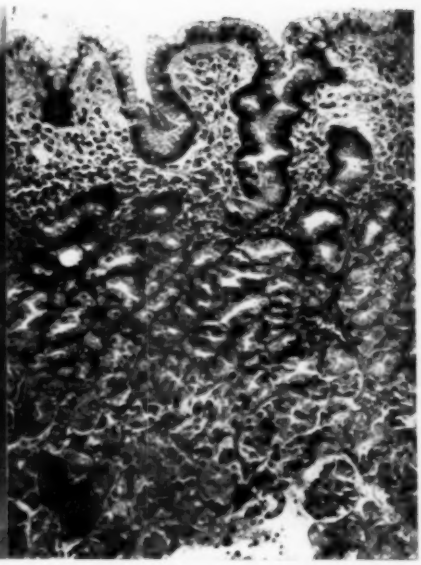

Fig. 6. Same case. Almost normal appearances, but foveolae lengthened and corkscrew-like. Some round cell infiltration and edema below surface.

Case 2 (1117/52). Female, aged 42; one year's epigastric distress. Clinical examination: N. A. D. Histamine test-meal: achlorhydria. *Roentgen examination of stomach:* Normal shape and size; rugae smooth and continuous (Fig. 3). *Gastroscopy:* Atrophy and reddening with edema of antrum. Atrophy and local superficial gastritis could also be seen in body of stomach. *Histology:* Only slight alterations present in surface epithelium. Foveolae lengthened and only small groups of normal glands noted. Throughout the mucosa moderate and occasionally marked round cell infiltration. Edema present beneath surface epithelium. Moderate alterations (thickening, irregularity, and lymphocyte infiltration) in muscularis mucosae (Fig. 4).

The report of one case with slight changes in the relief pattern, in which superficial gastritis was established both gastroscopically and histologically, now follows.

Case 3 (1062/52). Female, aged 50, with anxiety state and slight hypochromic anemia. Histamine test-meal: free HCl. *Roentgen examination of stomach:* Normal shape and size with almost continuous and slightly thickened folds. Residual content slightly increased (Fig. 5). *Gastroscopy:* Signs of superficial gastritis in antrum. The same changes occurred in the aboral region and minor curvature of body partly outside the correlation areas. *Histology:* Surface epithelium and body glands almost normal. Some of foveolae lengthened and corkscrew-like. Edema and slight round cell infiltration beneath surface epithelium (Fig. 6).

Fig. 7. Case 4. Mucosal folds irregular, coarse, thickened, and rigid ('tumour-forming').

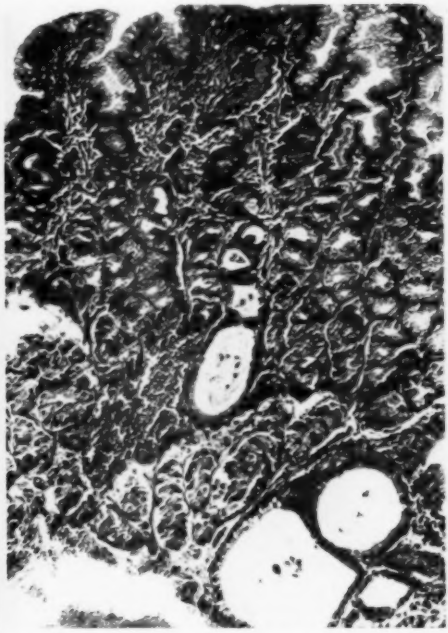

Fig. 8. Same case. Mucosa thickened with slight hyperplastic changes in epithelium. Foveolae lengthened. Large cysts at base.

The reports are concluded with two cases in which marked changes in the gastric mucosal pattern were observed roentgenologically and confirmed both gastroscopically and histologically.

Case 4 (1167/52). Male, aged 20, with epigastric distress and vomiting for about a week. Clinical examination: N. A. D. Histamine test-meal: free HCl. *Roentgen examination of stomach:* Tonus of gastric wall increased; folds irregular, coarse, thickened and rigid ('tumour-forming') (Fig. 7). *Gastroscopy:* Distinct hypertrophic changes in antrum and aboral and in middle parts of body. In some places the folds appeared enlarged and rigid. *Histology:* Some parts of mucosa obviously thickened. Slight hyperplastic changes in surface epithelium. Many foveolae distinctly lengthened. Scattered inflammatory foci present throughout mucosa. Groups of pseudopyloric glands and large cysts observed at base of mucosa (Fig. 8).

Case 5 (1184/52). Male, aged 36, with postprandial pain of one year's duration. Clinical examination: considerable epigastric tenderness on palpation. Histamine test-meal: free HCl. *Roentgen examination of stomach:* Normal size with increased motility. Folds irregular, coarse, thickened, and somewhat rigid (Fig. 9). *Gastroscopy:* Antrum

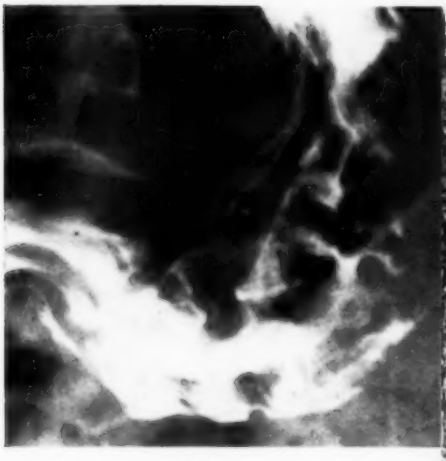

Fig. 9

Fig. 9. Case 5. Irregular, coarse, thickened folds of gastric mucosa.

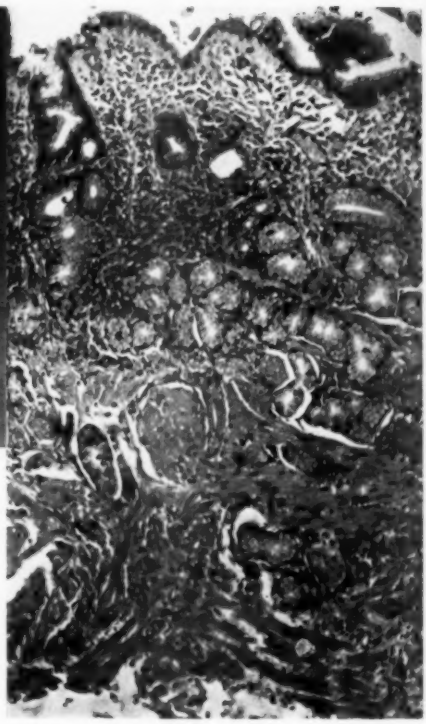

Fig. 10. Same case. Mucosa considerably thickened with hyperplastic changes in epithelium. Round cell infiltration and edema in mucosa. Muscularis mucosae thickened and split up.

Fig. 10

normal. Patchy superficial gastritis present in body of stomach within correlation area. *Histology:* Mucosa considerably thickened. Hyperplastic changes present in surface epithelium. No normal body glands found in body of stomach; most of glands pseudopyloric and often penetrating into muscularis mucosae. Moderate inflammatory cell infiltration throughout mucosa. Muscularis mucosae thickened, irregularly split up and slightly infiltrated with round cells (Fig. 10).

Discussion

Such roentgenologic changes as increased turgor, irregularity and coarseness of the mucosal folds, when occurring separately, appear to be relatively unimportant in the diagnosis of gastritis as verified only by gastroscopy, a method, which, as stated previously, is always subjective and far from ideal. It is true that these changes were observed much more frequently in the hypertrophic and superficial gastric mucosa groups than in the other groups, but only in part of the whole material.

Thickening of the folds, however, suggests, at least to some extent, hypertrophic and superficial gastritis.

On the other hand, the correlation of the roentgenologic changes in the mucosal pattern with the histologic findings was more positive. Thickening of the folds, especially, occurred remarkably often in subjects with hypertrophic alterations in the mucosa or with signs indicating superficial gastritis. Thickening of the folds, however, were present even in histologically normal gastric mucosa, yet there appears to be some justification for stating that thickening generally suggests hypertrophic and/or superficial alterations in the mucosa. This opinion differs somewhat from that expressed, for example, by SCHINDLER (14) who points out that thickness or thinness alone, as seen on roentgen examination, is meaningless and must not be interpreted as a sign of gastritis. No signs were found, however, in the mucosal relief characteristic of atrophic mucosa and this agrees well with the statements of previous authors (2, 3, 19). If, however, mucosal hyperplasia, superficial inflammation, and especially, gross changes in the muscularis mucosae, are associated with atrophy, they may cause marked changes in the relief pattern.

Where considerable alterations in the mucosal relief pattern occurred, there is, according to the present material, certainly a question of a pathologic gastric mucosa.

Certain conclusions may be drawn from the present investigation. It is evident that the roentgenologic relief pattern does not only reflect a functional state, but is also dependent upon the pathoanatomic condition of the gastric mucosa, particularly the muscularis mucosae, and upon the presence of mucosal hyperplasia or superficial inflammation. It is also evident that marked roentgenologic signs have a certain significance in the diagnosis of some types of gastritis. Only a minor number can, however, be diagnosed roentgenologically and as previously mentioned, the large and important group of atrophic gastritis does not, with some exceptional cases, come within the diagnostic scope of this examination method. In general, the results of correlation must therefore be considered far from satisfactory unless we try to avoid the most common sources of error and thus secure a reliable pathoanatomic diagnosis. We are convinced that with the roentgen technique we have used it is scarcely possible to reach a better correlation in cases in which the patho-anatomic changes are slight or even moderate. The examination of the stomach by means of a barium meal is actually a rather rough and superficial method for the purpose of diagnosing gastritis. It is derived from some twenty years ago and during the intervening long period no significant progress has been achieved in this field of roentgenology.

SUMMARY

An investigation was carried out to determine whether there are any roentgenologic signs which agree with the gastroscopic and histologic picture of the gastric mucosa. Four hundred and one (401) subjects were examined gastroscopically and roentgenologically, and in 203, biopsy specimens were obtained. None of the changes in the mucosal pattern, when occurring separately, was found to be of significance in the diagnosis of gastritis. In cases with marked changes, however, histologically detectable gastritis was always present.

ZUSAMMENFASSUNG

Eine Untersuchung ist vorgenommen worden, um festzustellen, ob es röntgenologische Merkmale gibt, die mit dem gastroskopischen und histologischen Bild der Magenschleimhaut übereinstimmen. 401 Personen wurden gastroskopisch und röntgenologisch untersucht und bei 203 Personen wurden Probeexzisionen vorgenommen. Keine der Veränderungen des Schleimhautbildes erwiesen sich, falls sie separat auftreten, als signifikativ für die Diagnose der Gastritis. In Fällen mit ausgesprochenen Veränderungen war jedoch histologisch nachweisbare Gastritis immer vorhanden.

RÉSUMÉ

Les auteurs ont cherché s'il existe des signes radiologiques qui concordent avec les aspects gastroscopiques et histologiques de la muqueuse gastrique. Ils ont examiné 401 sujets gastroscopiquement et radiologiquement et, sur 203 ils ont prélevé des fragments biopsiques. Aucune des modifications du relief muqueux, quand elle apparaît isolément, ne s'est montrée valable pour le diagnostic de gastrite. Cependant, dans les cas où les modifications étaient marquées, il existait toujours une gastrite détectable par l'histologie.

REFERENCES

1. Åkerlund, Å.: The roentgen diagnosis of ulcus duodeni with respect to the local "direct" roentgen symptoms. Acta radiol. 2 (1923), 14.
2. Ansprenger, A. and Kirklin, R. B.: Roentgenologic aspects of chronic gastritis. Critical analysis. Am. J. Roentgenol. 38 (1937), 533.
3. Benedict, E. B.: Correlation of gastroscopic, roentgenologic and pathologic findings in diseases of the stomach; an analysis of 245 proved cases. Am. J. Roentgenol. 55 (1946), 241.
4. Berg, H. H.: Röntgenuntersuchungen am Innenrelief des Verdauungskanals. 2. Auflage. Georg Thieme, Leipzig 1931.
5. Forssell, G.: Studies of the mechanism of movement of the mucous membrane of the digestive tract. Am. J. Roentgenol. 10 (1923), 87.
6. Kennedy, T. J.: Difficulties in the differential diagnosis of gastric carcinoma and gastritis. Surgical considerations. Radiology 59 (1952), 367.
7. Kirklin, R. B.: Roentgenologic aspects of chronic gastritis. Proc. Staff. Meet. Mayo Clin. 14 (1939), 59.
8. Konjetzny, G. E.: Die Entzündungen des Magens. *In* Henke-Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie IV/2 (1928), 768.

9. Mc Glone, F. B.: Clinical aspects of gastritis and the gastroscopical findings. Radiology 59 (1952), 358.
10. Palmer, E. D.: Histology of the normal gastric mucosa. Gastroenterology 21 (1952), 12.
11. Rendich, R. A.: The roentgenographic study of the mucosa in normal and pathological states. Am. J. Roentgenol. 10 (1923), 526.
12. Renshaw, R. J. F.: Correlation of roentgenologic and gastroscopic findings from the standpoint of the gastroenterologist. Am. J. Roentgenol. 51 (1944), 585.
13. Schindler, R.: Gastroscopy: The endoscopic study of gastric pathology. The University of Chicago Press, Chicago 1950.
14. — Gastritis. Grune & Stratton, London 1947.
15. Schindler, R., Necheles, H. and Gold, R.: Surgical gastritis. Surg., Gynec. & Obst. 69 (1939), 281.
16. Schindler, R. and Templeton, F.: Comparison of gastroscopic and roentgen findings. Radiology 29 (1937), 472.
17. Setälä, K., Ermala, P. and Siurala, M.: An instrument for gastric biopsy of the Rhesus monkey. J. Nat. Cancer Inst. 14 (1953), 141.
18. Stampfli, W. P.: Roentgenological exploration of gastric rugae with reference to the diagnosis of gastritis. Radiology 59 (1952), 336.
19. Templeton, F.: X-ray examination of the stomach. University of Chicago Press, Chicago 1944.
20. Walk, L.: The roentgen signs of gastritis. Am. J. Roentgenol. 60 (1948), 77.
21. Vaughan, W. W., Gunter, J. U. and Erwin, E. A. Jr.: Enlarged gastric rugae. A correlation of the roentgenologic, gastroscopic, pathologic and clinical findings. Radiology 56 (1951), 813.
22. Ylvisaker, R. S., Carey, J. B. Sr., Myhre, J. and Carey, J. B. Jr.: Biopsy studies of the gastric mucosa. Gastroenterology 28 (1955), 88.

FROM THE ROENTGEN DEPARTMENT (DIRECTOR: PROF. FOLKE KNUTSSON), UNIVERSITY HOSPITAL, UPSALA, SWEDEN

SPONTANEOUS PERFORATION OF THE OESOPHAGUS

by

Gunnar Tilling

The term 'spontaneous perforation of the oesophagus' is used to denote sudden rupture of that organ in an apparently healthy subject. It is a rare event, but must be borne in mind in the differential diagnosis of acute conditions of the abdomen and thorax. Most cases are diagnosed only at post-mortem examination, and few have been described in the literature.

Case histories

Case 1. Male, aged 54 years, previously healthy and with no history of peptic ulcer, suddenly developed nausea and, during the act of vomiting, experienced sudden severe pain between the scapulae. Shortly afterwards the pain moved to the left hypochondrium. On admission a tentative diagnosis of perforated peptic ulcer was made. Routine roentgenography, however, revealed no free gas in the abdomen but showed a left-sided hydropneumothorax with a considerable amount of fluid and a little gas (Fig. 1). Perforation of the oesophagus was therefore suspected, and its examination with a contrast medium disclosed a perforation to the left of the lower part of the oesophagus into the left pleural cavity (Fig. 2). The patient was in severe pain, with a normal temperature, the pulse rate being 120. Left thoracotomy (resection of 8th rib) was performed 4 hours after admission and 8 hours following the attack. A perforation of the oesophageal wall admitting a finger tip was found on the left side, 4 cm above the diaphragm. The edges of the lesion were quite soft. No ulcer or tumour was visible, or could be demonstrated microscopically, only oesophagitis being present. The oesophagus was sutured.

The post-operative course was initially uneventful and the patient was even allowed up a little. Three weeks after operation, however, he started to develop signs of septicaemia, and died on the 24th day.

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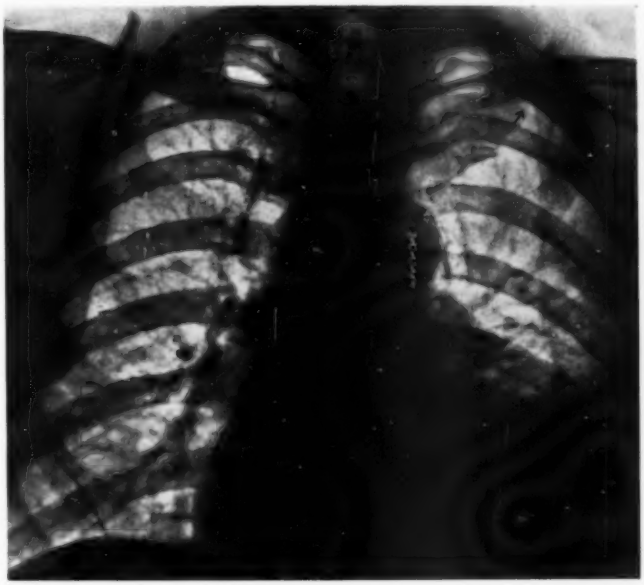

Fig. 1. Case 1. Hydropneumothorax on the left side.

Case 2. Male, aged 51 years, with a 16-year history of peptic ulcer. During the act of defaecation he suddenly experienced a sensation of 'something giving way' in the chest. He became dyspnoeic, vomited, and experienced very severe pain. He was sent to hospital as a case of coronary occlusion, and was treated under this diagnosis for 24 hours. Roentgenography of the chest ('ward mobile') then revealed massive pleural effusion on the left side with marked mediastinal displacement and mediastinal emphysema (Fig. 3). Six hundred (600) ml of gas and 2,000 ml of blackish-brown fluid were aspirated. The condition of the patient rapidly deteriorated, and he died a few hours later, one day after the onset of symptoms. Necropsy report: perforated peptic ulcer in the posterior oesophageal wall immediately above the cardia and opening directly into the left pleural cavity. Scarring following peptic ulcer in the pyloric region of the stomach.

Case 3. Male, aged 49 years, with a 15-year history of peptic ulcer, admitted to hospital for treatment of the old condition, an ulcer of the duodenum being diagnosed. While in hospital he developed sudden severe pain in the chest. The blood-pressure was low, and the electrocardiogram indicated coronary insufficiency. He was treated under the diagnosis of coronary occlusion. Roentgenography of the chest ('ward mobile') 4 days after the acute attack revealed considerable opacity of the left lung field. The patient died soon afterwards. At necropsy foul-smelling gas escaped from the left pleural cavity, which also contained 1,500 ml of stinking, greyish-green fluid. The lower end of the oesophagus showed a perforation in the form of a slit opening posteriorly into the left pleural cavity. No tumour was found, but an abscess was present in the mediastinum.

Fig. 2. Case 1. Perforation of the oesophagus above the diaphragm into the left pleural cavity.

According to ROBB-SMITH, the total number of cases published up to 1940 was 40. In 1946 the figure had risen to 50 (BARRETT). The condition began to be treated operatively in 1947, and by 1949 16 cases had been diagnosed during life. The total number of successful operations reported up to 1953 was 23. In 1955 BORRIE described three operated cases and BOULLE one case.

In most cases the oesophagus ruptures at the distal end just above the cardia, the perforation being commonly posterior and to the left. Many give a history of peptic ulcer. Peptic ulcer of the oesophagus is not a rare condition, and is predisposed by one or both of the following factors (HELLMAN):

1. Islands of gastric mucosa in the oesophageal epithelium.
2. Regurgitation of stomach contents into the oesophagus.

In many cases it is impossible to demonstrate an ulcer in operation and post-mortem specimens. It would often seem unreasonable that a perfectly healthy oesophagus should simply tear, since the epithelium of this organ is as thick and strong as that of the skin, the muscle layers also being extremely powerful. It is probable that an ulcer or pre-ulcerous condition such as local oesophageal necrosis exists in these cases (STRÜMPELL-SEYFARTH).

The oesophagus may perforate into the mediastinum or one or both pleural cavities. Theoretically it could also rupture into the aorta or pericardium.

Of the pleural cavities, the left is more commonly involved than the right. There is a plausible anatomic explanation for this. The relations of

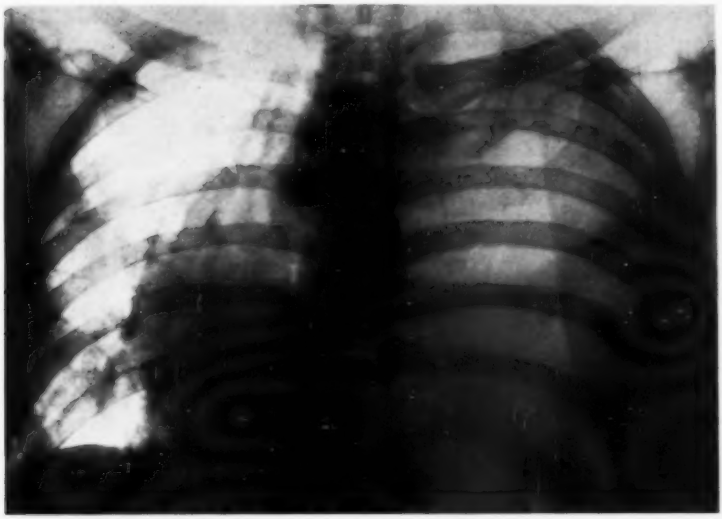

Fig. 3. Case 2. Hydropneumothorax on left side with marked mediastinal displacement to right. Mediastinal emphysema. ('Ward mobile' film.)

the pleura in the posterior mediastinum have been described by KNUTSSON. The left pleura follows the lateral borders of the vertebral bodies as far as Th 12. The right pleura, however, forms a deep pouch between the levels of the 5th and 10th thoracic vertebrae and extending beyond the midline, the two pleurae thus lying very close together in this region. Between Th 10 and Th 12 the right pleura follows a diagonal course out to the right lateral border of Th 12. Approached from the dorsum, removal of the vertebral bodies reveals the great vessels (aorta, vena azygos, ductus thoracicus); anterior to these lie the mediastinal edges of the pleural cavities, with the deep pouch formed by the right pleura: inferiorly lies the oesophagus, which at this level curves over to the left of the midline. The lower end of the oesophagus is seen to have no relation with the right pleura, but lies in close contact with the left. For this reason rupture of the lower end of the oesophagus commonly takes place into the left pleural cavity, whereas the upper parts of the organ more readily perforate into the right.

Diagnosis. Spontaneous rupture of the oesophagus constitutes an acute emergency and occurs almost exclusively in connexion with vomiting or defaecation. The patient is shocked, with severe pain in the chest and upper abdomen, but the clinical picture varies according to the site of the lesion and the direction in which the perforation takes place. For

example, perforation into the mediastinum may produce local emphysema spreading upwards to the cervical region. The most significant radiologic sign, however, is the presence of hydropneumothorax. When found in a patient with acute severe pain in the thorax and/or upper abdomen it should always lead to the suspicion of rupture of the oesophagus, and examination of the latter should be carried out with a sterile absorbable medium as contrast agent.

The *differential diagnosis* includes perforation of a peptic ulcer, coronary occlusion and pulmonary embolus. Acute cholecystitis and acute pancreatitis should also be excluded.

SUMMARY

Three cases of spontaneous perforation of the lower end of the oesophagus into the left pleural cavity causing hydropneumothorax, are described. Two of the cases were diagnosed clinically as coronary thrombosis and one as perforated peptic ulceration.

ZUSAMMENFASSUNG

Drei Fälle mit spontaner Perforation des unteren Abschnittes des Ösophagus in die linke Pleurahöhle, wodurch ein Hydropneumothorax entstand, werden beschrieben. In zwei der Fälle war die klinische Diagnose Coronarthrombose und im dritten Falle perforiertes peptisches Geschwür.

RÉSUMÉ

L'auteur présente trois cas de perforation spontanée de l'extrémité inférieure de l'œsophage dans la cavité pleurale gauche, causant un hydropneumothorax. Le diagnostic clinique était dans deux cas celui de thrombose coronaire et dans un cas celui d'ulcère peptique perforé.

REFERENCES

Barret, N. R.: Spontaneous rupture of the oesophagus. Thorax 1 (1946), 48.
— Report of a case of spontaneous perforation of the oesophagus successfully treated by operation. Brit. J. Surg. 35 (1947), 216.
Borrie, J.: Spontaneous rupture of oesophagus. Brit. M. J. 1955, p. 23.
Boulle, J. R.: Spontaneous rupture of oesophagus. Brit. M. J. 1955, p. 25.
Hellman, J.: Das Ulcus pepticum oesophagi. Bruns Beitr. 115 (1919), 449.
Knutsson, F.: The mediastinal pleura. Acta radiol. 43 (1955), 265.
Strümpell-Seyfarth: Lehrbuch der speciellen Pathologie und Therapie, 31/32 Aufl., S. 627.

FROM THE ROENTGEN DEPARTMENT (DIRECTOR: F. DALITH), GOVERNMENT HOSPITAL, TEL-HASHOMER, ISRAEL

SYSTOLIC EXPANSION OR AORTO-DIASTOLIC DISPLACEMENT

A roentgenkymographic study of left atrial movements in mitral cardiopathy

by

F. Dalith

Systolic expansion of the left atrium has long been recognized as a significant sign in the radiologic diagnosis of mitral regurgitation. However, the reliability and even the very nature of this phenomenon have become doubtful with the appearance of a new criterion of mitral insufficiency — the palpation of a regurgitant jet by the exploring finger of the surgeon. A re-evaluation of 'systolic expansion' is, therefore pertinent and timely.

Review of the literature. 'Systolic expansion of the left atrium' refers to the outward movement of the left atrial borders synchronous with the inward movement of the ventricular borders. ROESLER (13) describes the phenomenon as follows: "When the extension of the left atrium into the right lung field partly overlaps the right atrial border, a peculiar appearance can be noted in a good many instances: the cranial and the caudal contours alternate in movement, so that a see-saw action results. The lateral movement of the cranial (left atrial) contour is simultaneous with the ventricular systole. (This has been verified by kymography.)" These pulsations may also be observed on the barium-filled esophagus outlining the left atrial wall. 'Systolic expansion' may be noted fluoroscopically and registered by graphic methods (roentgenkymography, electrokymography, or esophageal pulse record).

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The pulsatory phenomenon under discussion is observed in a good many cases of mitral cardiopathy, but not in all, and not always to the same degree. ELKIN et coll. (4) consider that the type and degree of left auricular movement is determined by many factors which include the size of the left auricle, the distensibility of its wall, the presence of mural thrombus, auricular fibrillation, the rate of filling from pulmonary veins, transmitted thrust from the right auricle in tricuspid incompetence, and systolic contraction of a hypertrophied right ventricle. DAVISON & EPPS (3) add to this list the degree of mobility of the antero-medial cusp of the mitral valve and bodily movement of the heart as a whole.

As to the hemodynamic explanation of the phenomenon there has been, until now, no complete agreement in the literature. ROESLER (13) assumes that systolic expansion is the expression of the reflux of blood from the left ventricle into the left atrium during ventricular systole due to incompetency of the mitral valve. This explanation is generally accepted today by the majority of authors. ZDANSKY (16), however, emphasizes that, even under normal conditions, the diastolic inflow into the left atrium during ventricular systole causes a sudden dilatation of this chamber. According to ZDANSKY, systolic expansion of the left atrium, as observed in cases of mitral cardiopathy, is at least in part the result of the sudden inflow of blood from the hyperemic pulmonary venous system. FROMENT *et coll.* (5) attribute the abnormal left auricular movement to paradoxical retropulsion of the auriculo-ventricular septum into the left auricle during ventricular systole. LEMKE (8) expresses doubt that the relatively small quantity of regurgitant blood in cases of mitral incompetency is sufficient to abolish the normal inward movement of the left atrial wall during the isometric contraction of the left ventricle; even greater the doubt that this small quantity may be able to convert it to an outward movement.

The majority of authors today concludes that expansile pulsation of the left atrium is not significant for mitral regurgitation and that there is need for caution in its evaluation. The absence of the sign does not rule out incompetence of the valve, as observations on patients with marked and predominant regurgitation have shown (ABELMAN, ELLIS & HARKEN (1), LOGAN & TURNER (9), DAVISON & EPPS (3). On the other hand, the sign has also been observed in patients with pure mitral stenosis in whom no regurgitant jet was felt at operation (ELKIN *et coll.* (4), DAVISON & EPPS (3) and FROMENT *et coll.* (5)), and even in people with apparently normal hearts (LOGAN & TURNER (10)). In view of these observations LOGAN & TURNER (9) and WOOD (15) conclude that only severe systolic expansion with certainty suggests the predominance of mitral incompetence. Reviewing their experience with 'systolic expansion' and evaluating roentgen slit kymography, LEHMANN & CURRY (7) state that this method

is of little aid in the detection of mitral insufficiency in any individual case. Similar conclusions with regards to esophageal pulse records and left auricular electrokymograms are reported by ZOOB (17) and DAVISON & EPPS (3).

Author's investigations

Our own observations on patients with mitral cardiopathy demonstrating 'systolic expansion' aroused similar doubts as to the reliability of the sign. The discrepancy between the generally accepted assumption as to the nature of the phenomenon ('auricular distension by regurgitant blood stream from the ventricle') and the findings during operation ('no regurgitant jet') provided the incentive for the present kymographic study.

Material. The study is based on 64 kymographic examinations in a series of 131 cases of mitral stenosis which were operated upon by Y. M. PAUZNER. The kymograms are mainly of patients in whom 'systolic expansion of the left atrium' was observed preoperatively by the radiologist and 'pure mitral stenosis without regurgitant jet' was established by the surgeon by intracardiac palpation. The remainder of the material consists of cases in which no 'systolic expansion' was observed and of cases in which a 'regurgitant jet' was palpated. Adequate comparisons of possible combinations are therefore available.

Method. An a. p. kymogram was made routinely in every patient. When systolic expansion was observed during fluoroscopy, one or more kymograms in oblique projections were added. Barium paste was given in order to outline the wall of the left atrium. The exposure conditions of the a. p. kymograms were such that the esophagus and the aorta were discernible in the heart shadow and that optimal visualization of the heart borders on both sides was obtained. For demonstration of the aortic pulsations the 10° LAO position was found to be very useful.

Four areas were investigated in the kymogram (Fig. 1):

The aortic knob area (Area A), the left atrial area (Area B), the ventricular area (Area C), the subdiaphragmatic area (Area D).

The pulsations of the esophagus were compared with those of the atrial and ventricular borders of the heart in the same area and with those of the left border of the aorta

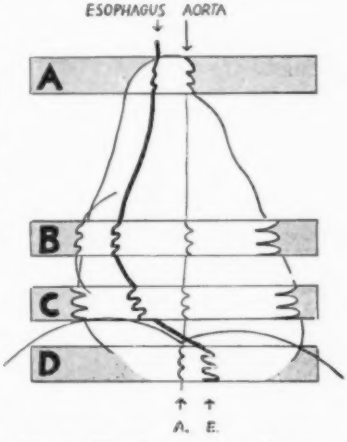

Fig. 1. The four areas of kymographic investigation: A: The aortic knob area. B: The left atrial area. C: The ventricular area. D: The subdiaphragmatic area.

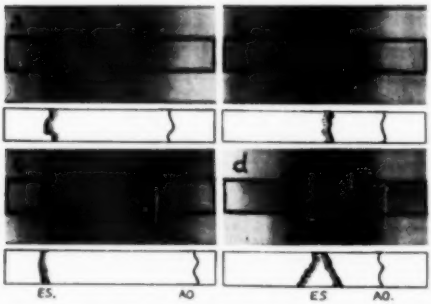

Fig. 2. The direction of the esophageal pulsations in area B in cases of pure mitral stenosis: a. Esophagus displaced to right — esophageal movements directed to right; b. Esophagus displaced to right — no definite direction of esophageal movements; c. Esophagus in midline — esophageal movements directed to left; d. Esophagus in midline — bi-directional esophageal movements.

in all four areas. Direction, shape, and amplitude of the pulsations were determined.

The material is divided into three groups according to the nature of the valvular lesions: I. Pure mitral stenosis (without regurgitant jet); II. Predominant mitral stenosis (with regurgitant jet) and III. Predominant mitral regurgitation.

It should be emphasized that the diagnosis is that which was established by the surgeon at the time of operation.

Observations

Group I, *pure mitral stenosis* (without regurgitant jet) consists of 55 cases.

The waves of the esophagus in the left atrial area (*B*). The *direction* of the esophageal pulsations as observed in the left atrial area (B) in the a. p. kymogram is found to be dependent on the course of the esophagus.

When the esophagus is displaced to the right, the esophageal movements are either directed to the right (Fig. 2 a) or no definite direction can be discerned (Fig. 2 b).

In cases with midline position of the esophagus four variants are observed: the pulsations of the esophagus are directed to the right or to the left (Fig. 2 c), they are bi-directional (Fig. 2 d), or no definite direction can be determined.

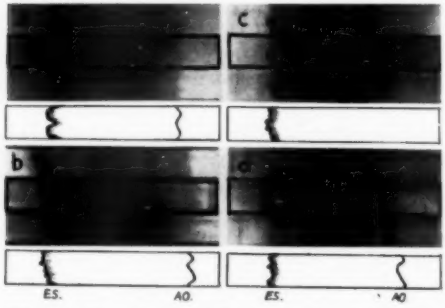

Fig. 3. The shape and amplitude of the esophageal pulsations in area B in cases of pure mitral stenosis: a. Large type; b. Small type; c and d. Mixed type.

The shape and amplitude of the esophageal pulsations are of three types: *Large type:* The waves are identical or similar in shape and amplitude to the pulsations of the great vessels (aorta and pulmonary artery). The duration of one wave of this type corresponds to the duration of one ventricular wave (Fig. 3 a). *Small type:* About three waves of this type corresponds to one ventricular wave

(Fig. 3 b). *Mixed type:* An analysis of these pulsations reveals them to consist of the two above-described forms, one superimposed on the other (Fig. 3 c & d).

The waves of the esophagus in the left atrial area (*B*) as compared with *the esophageal waves in the ventricular area* (*C*) are always identical in direction and for the most part similar in shape and amplitude.

The waves of the esophagus as compared with those of the borders of the heart and aorta. The esophageal pulsations were compared with those of the border of heart and aorta. For this purpose those kymograms which show the large type of pulsations were chosen for the following reasons: The small type of waves, as clarified by electrokymographic studies of ANDERSON (2) and SCHLEGEL & SCHOELMERISCH (14), represents, in cases with sinus rhythm, the intrinsic pulsations of the left atrium. These pulsations, however, are too small in amplitude and too short in duration to be perceived by the fluoroscopist's eyes. This type therefore, was felt not to be useful for the present study. The mixed type, on the other hand, was excluded on account of its complexity.

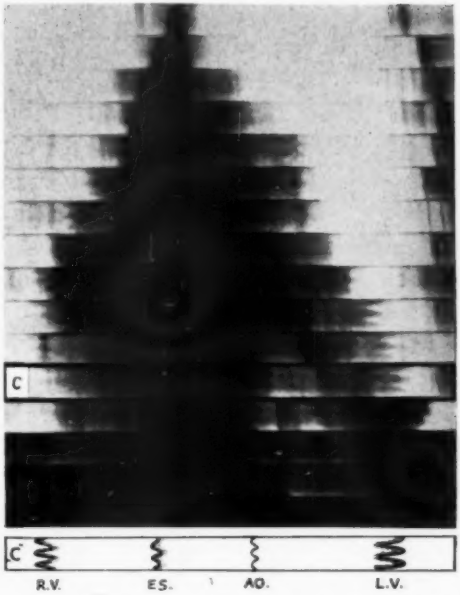

Fig. 4. Pure mitral stenosis: the direction of the esophageal pulsations in C area is identical with those of the left ventricular border, but reversed as compared to those of the right ventricular border.

The right border of the left atrium (*Area B*). In all cases showing displacement of the esophagus to the right and in some cases with midline position of the esophagus, the esophageal movements in the left atrial area (B) are identical in direction and in shape as compared with those of the right heart border in the same area, provided that the right border of the left atrium extends into the right lung field. In the remainder of cases with midline course of the esophagus the esophageal pulsations are oppositely directed to those of the right border of the left atrium.

Borders of ventricles (*Area C*). Comparison of the esophageal waves in the area B with those of the borders of the ventricles in the area C shows that in cases with esophageal displacement to the right and in some ca-

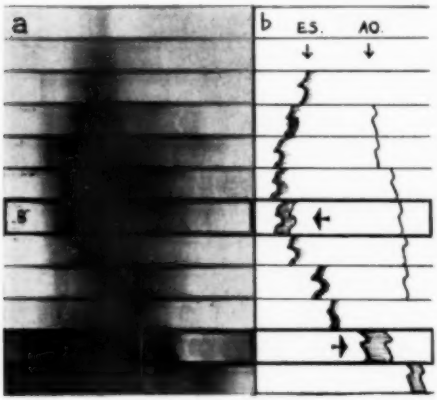

Fig. 5. a. Pure mitral stenosis: the direction of the esophageal pulsations is opposite to those of the aorta (left border) in area A and in the areas B and C (thoracic part of descending aorta), but identical in area D. b. Diagrammatic sketch of a.

ses with midline position the direction of the esophageal movements is identical with those of the left heart border, but reversed as compared with the right (Fig. 4).

In the remainder of cases with midline course of the esophagus, the direction of the esophageal pulsations is identical with that of the right heart border and opposite to that of the left.

Aorta (All four areas): In all cases with displacement of the esophagus to the right and in some cases with midline position the pulsations of the esophagus, as observed in the area B (left atrium), compared with those of the left border of the aorta in the area A (knob) and B and C (thoracic part of descending aorta), are identical in shape, but opposite in direction. The pulsations of the esophagus in the area D (subdiaphragmatic area), however, are identical in shape and direction as compared with those of the left border of aorta in all four areas (Fig. 5).

In the remainder of cases with midline position of the esophagus, the esophageal pulsations, as observed in the area B compared with the left border of the aorta in all four areas are identical in shape and direction.

Group II, *predominant mitral stenosis* (with regurgitant jet), consists of 6 cases.

The direction of the esophageal pulsations and their patterns in concomitant mitral regurgitation were identical with those observed in pure mitral stenosis (Fig. 6).

Group III, *predominant mitral regurgitation*, consists of 2 cases.

In two patients, who were submitted to surgery with the preoperative diagnosis of predominant mitral stenosis, predominant regurgitation was established at operation by the surgeon. The kymogram of one patient of this group showed midline position of the esophagus and waves of the large type, directed to the right before the operation (Fig. 7 a). At the follow-up examination, about six months after the operation, no change of direction and pattern was observed, but the esophagus was displaced to the right (Fig. 7 b).

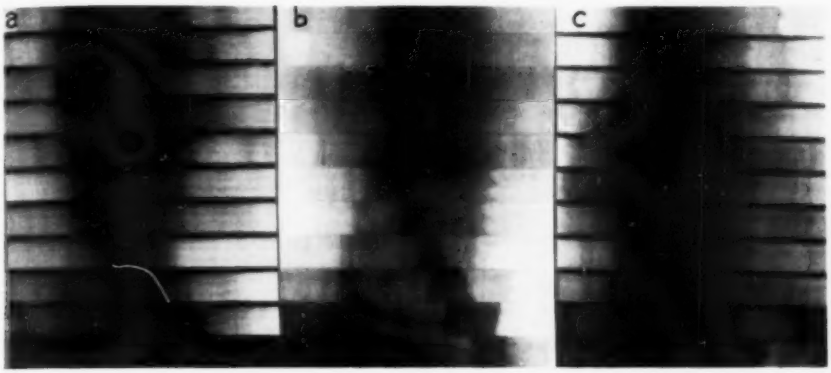

Fig. 6. The direction and pattern of the esophageal pulsations in cases of predominant mitral stenosis (with regurgitant jet): a. Esophagus displaced to the right — esophageal movements directed to right; b. Esophagus in midline — no pulsations; c. Esophagus in midline — esophageal movements directed to left. The pulsations are identical with those observed in pure mitral stenosis.

In the second patient, the esophagus was displaced to the right and pulsations of the large type, directed to the right, were also seen (Fig. 7 c).

It was impossible to establish any difference between the pattern of the left atrial movements in predominant mitral incompetence and of those in pure mitral stenosis.

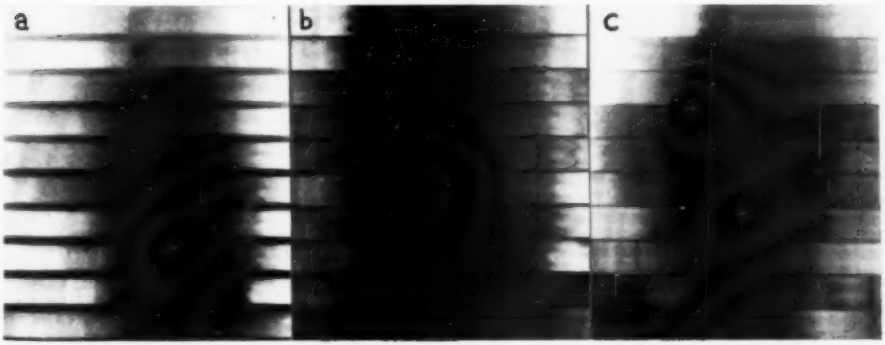

Fig. 7. The direction and pattern of the esophageal pulsations in cases with predominant mitral regurgitation: a. Esophagus in midline — movements directed to right; b. The previous patient, six months after operation; c. Esophagus displaced to the right — movements directed to right. The pulsations are identical with those observed in pure mitral stenosis.

The findings of the 64 kymographic examinations of this series are summarized in Table 1.

Table 1

The findings in 64 kymographic examinations on patients with mitral cardiopathy as observed on the barium-filled esophagus in the left atrial area

	Surgical diagnosis	Number of examinations	Esophageal pulsations in the left atrial area							
			Direction				Pattern			
			Right	Left	Bi-dir.	Undet.	Large	Mixed	Small	Undet.
Displacement of the esophagus to the right (38 examinations)	Pure mitr. sten.	32	26	—	—	6	7	18	1	6
	Predominant mitr. sten.	4	4	—	—	—	2	2	—	—
	Predominant mitr. incomp.	2	2	—	—	—	1	1	—	—
Midline-position of the esophagus (26 examinations	Pure mitr. sten.	23	7	4	4	8	9	6	—	8
	Predominant mitr. sten.	2	—	1	—	1	—	1	—	1
	Predominant mitr. incomp.	1	—	—	—	—	1	—	—	—

The author's observations may be summarized as follows.

1. The left atrial movements in patients with pure mitral stenosis as registered kymographically on the barium-filled esophagus were compared with the movements in patients with predominant mitral stenosis (with associated mitral incompetence) and with predominant mitral incompetence. No essential difference in the pattern of the esophageal movements could be established between these three types of mitral valvular lesions.

2. In cases with mitral incompetence (concomitant as well as predominant) the author never observed a kymographic equivalent for systolic expansion of the left atrium, *i. e.* an accentuated outward movement during the early systolic phase of isometric ventricular contraction.

3. The analysis of the esophageal waves in mitral valvular lesions shows that their shape is identical with or similar to the waves of the thoracic aorta and that their direction is dependent upon the positional relationship of the esophagus to the aorta.

Discussion

Nature and genesis of the pulsations. The identity in shape of the esophageal pulsations in patients with mitral cardiopathy with those of the aorta and the dependency of their direction upon the relative position

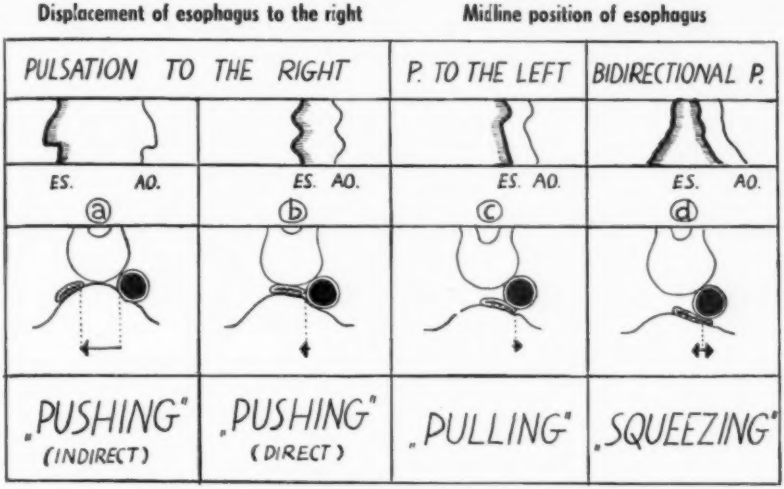

Fig. 8. The production of varying patterns of esophageal pulsations depending upon the relation of the esophagus to the aorta.

of the esophagus to the aorta make it probable that these pulsations originate in the aorta. The author supposes that the fluoroscopically observed outward movements of the right heart border (left atrium) and of the barium-filled esophagus are due to the diastolic expansile thrust of the aorta, which occurs synchronously with the ventricular systole, and not, as generally assumed, due to expansion of the left atrium caused by a regurgitant blood stream. The phenomenon of the so-called 'systolic expansion' is thought to be due to pulsatory displacement (change of position) of the left atrium rather than to expansion (change of volume). The movements are mainly extrinsic pulsations and the designation 'aorto-diastolic displacement' is proposed.

The shape of these waves depends upon whether intrinsic waves are present or not. In the presence of intrinsic waves, we observe the mixed type of pulsations (the small waves superimposed on the large); in the absence of intrinsic pulsations, the large type is noted.

As to the direction of the pulsations four groups are distinguished in this series:

a. Pulsations directed to the right are observed in cases with displacement of the esophagus to the right (indirect transmission of the pulsatory movements of the right aortic wall through the distended left atrium) (Fig. 8 a), and in cases with midline course of the esophagus beside the aorta (direct transmission) (Fig. 8 b).

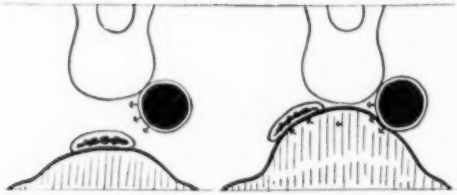

Fig. 9. Schema demonstrating the genesis of the esophageal pulsations in mitral cardiopathy: a. In the normal there is no contact between the aorta on one hand and the left atrium and the adjacent esophagus on the other; b. In mitral valvular disease the enlarged left atrium establishes direct contact with the aorta and serves to bridge the gap between the aorta and the esophagus. In this way the engorged left atrium mediates the transmission of aortic pulsations to the esophagus.

b. Pulsations directed to the left are seen in cases with midline position of the esophagus in front of the aorta (pulsatory drawing of the adherent esophagus) (Fig. 8 c).

c. Bi-directional pulsations are established in some cases with midline position of the esophagus in front of the aorta. They are thought to be produced by a 'squeezing-effect': The barium-filled esophagus is squeezed between the left atrium from in front and the aorta from behind during the expansion of the latter (Fig. 8 d).

d. Pulsations with undeterminated direction or absence of pulsation may be noted, as the author supposes, in cases with minimal enlargement of the left atrium (no contact between left atrium and aorta), in cases with thickening of the atrial wall (for instance, marked fibrosis or calcification of the endocardium), or in cases in which the atrial cavity is occupied by thrombus.

Absence of pulsation in the a. p. direction may be apparent only and must be checked by examination in oblique positions.

The high incidence of the phenomenon in mitral valvular lesions may be explained as follows: In mitral cardiopathy the left atrium increases into the posterior mediastinum and comes in contact with the thoracic aorta (Fig. 9). The aorta, a relatively rigid pipe, prevents further expansion of the atrium to the left in much the same manner as the vertebral bodies inhibit further enlargement posteriorly. When intimate contact between the aorta and left atrium has been created, the great vessel, owing to the higher pressures prevailing, transmits its pulsations to the atrium. These extrinsic pulsations, transmitted to the adjacent esophagus, may be perceived by fluoroscopy and registered by graphic methods.

Concerning the different amplitude of the pulsations in different patients, the author agrees with the list of factors determining the degree of left auricular movements as presented by Elkin et coll. (4). Different amplitude is observed also in the same patient in different fluoroscopic positions.

The pulsations are usually observed more prominently in the RAO than in the a. p. position and in some instances they may be entirely absent in the latter and noted only in the oblique view (Fig. 10). How can we

Fig. 10. Variations in amplitude of the esophageal pulsations in cases with pure mitral stenosis in different radiographic positions: a. A. p.: small amplitude; b. (the same patient) RAO: large amplitude. c. A. p.: small amplitude. d. (the same patient) RAO: large amplitude.

explain this fact? The enlarged left atrium comes in contact laterally with the expanded right lung covered by the tense pleura. Latero-posteriorly, on the other hand, lies only the loose areolar tissue of the right posterior mediastinum. We suggest that the lateral movement of the atrium may be damped by the cushioning effect of the expanded lung, whilst the obliquely directed movements find no marked resistance in the loose areolar tissue of the posterior mediastinum. This assumption is felt to be supported by our investigations in the human cadaver. When examining the posterior mediastinum the exploring finger finds resistance to movement directed to the right against the mediastinal pleura, but is relatively unhindered in the oblique direction.

McKusick (11), in his study on mitral regurgitation, reports a case (Case III) with disappearance of 'systolic expansion' after valvotomy. In the preoperative stage McKusick observed, in a markedly enlarged heart, lateral movements of the esophagus during ventricular systole. Mitral regurgitation was therefore assumed. But, on palpation at operation, no regurgitant jet was found and commissurotomy was performed. Six months later, when the heart returned to normal size, expansile pulsations were no longer noted in the kymogram. McKusick concludes that it is difficult to reconcile the roentgen kymographic findings with the surgeon's impression. The present author, however, proposes the following explanation: During the preoperative stage there was intimate contact between the left atrium and the aorta due to enlargement of the heart chamber. After the commissurotomy, when the heart returned to normal size, the contact between atrium and aorta was lost and the aortic pulsations were no longer transmitted. This interesting case, in our opinion, supports the assumption that the atrial movements under discussion

are transmitted pulsations and not expansile. (In the series of the present study we did not observe a similar case.)

The appearance of the sign in other valvular lesions (as aortic insufficiency), in other heart diseases (tetralogy of Fallot), and even in healthy persons, is also assumed to be dependent upon the topographic relationship between esophagus and aorta in any individual case.

Regurgitant blood stream from the left ventricle. The author doubts that the movements of the large type could be the kymographic equivalent for change of volume due to a regurgitant blood stream from the left ventricle for two reasons: (1) Large waves are observed in patients with surgically proven pure mitral stenosis without regurgitant jet and (2) in no case of surgically proven regurgitant jet, apart from the wave during the phase of diastolic expansion of the aorta, was another wave noted kymographically that corresponded to the isometric ventricular contraction phase.

Inflow of blood from the pulmonary veins. According to ZDANSKY (16), the inflow of blood into the left atrium from the hyperemic pulmonary venous system during ventricular systole produces, even under normal conditions, a sudden dilatation of this chamber. However, the waves, created by increase of left atrial volume due to inflow of blood from the pulmonary veins are of the small type, as shown by SCHLEGEL & SCHOELMERISCH (14).

Displacement of the atrium due to retropulsion of the auriculo-ventricular septum. In those cases in which the cusps are not fibrotic or calcified, in other words, when a certain amount of elasticity of the valve apparatus remains, the surgeon (Y. M. PAUZNER) has been able to palpate a bulging of the broad anterior leaflet towards his finger with each ventricular systole. It has been the surgeon's impression, however, that the extent and force of this bulging would not be able to produce a fluoroscopically visible movement of the left atrial wall (12). On the other hand, if such should be the mechanism of 'systolic expansion', as suggested by FROMENT (5), it would be expected that the left atrial movements, as seen on the esophagus, would be similar in shape to the left ventricular waves. However, the identity in shape of the esophageal waves with the aortic rather than with the ventricular pulsation speaks in favour of pulsatory displacement by the great vessel and not the heart chamber.

Radiologic diagnosis of mitral regurgitation

The radiologic demonstration or exclusion of mitral regurgitation, associated with mitral stenosis, is the source of many difficulties to the radiologist today. According to the literature four radiologic signs are suggestive of mitral regurgitation: (1) Left ventricular enlargement, (2)

Rapid left ventricular filling wave, (3) Marked left atrial enlargement, and (4) Systolic expansion of the left atrium. However, the exact radiologic estimation of the size of the left ventricle is often difficult or impossible; moreover, marked regurgitation may exist without enlargement of this heart chamber. The rapid left ventricular filling wave (HIRSCH & GUBNER, 6) has not been proven to be a significant radiologic sign. Marked and even giant enlargement of the left atrium is not unusual in pure mitral stenosis and may be absent in mitral regurgitation. 'Systolic expansion' of the left atrium has lost its reliability. Thus, we must state, that for the present at least, the contribution of the radiologist in the prediction of mitral regurgitation is limited.

SUMMARY

The discrepancy between the generally accepted assumption as to the nature of 'systolic expansion of the left atrium' and the findings of the surgeon on intracardiac palpation ('no regurgitation jet') provided the incentive for a roentgenkymographic study on 131 patients with mitral cardiopathy.

The author concludes that the sign is caused by pulsatory displacement of the left atrium (during ventricular systole) due to the expanding aorta rather than, as generally assumed, by atrial expansion due to a regurgitant blood stream from the left ventricle. The designation 'aorto-diastolic displacement' is proposed for the phenomenon.

ZUSAMMENFASSUNG

Die Diskrepanz zwischen der allgemeinen anerkannten Ansicht bezüglich der Natur der »systolischen Expansion des linken Vorhofes« und der Befunde des Chirurgen bei intrakardialer Palpation (»kein Regurgitationsstrahl«) bildete das Motiv für eine röntgenkymographische Studie an 131 Patienten mit mitralen Herzkrankheiten.

Der Verfasser konkludiert, dass das Symptom eher durch eine pulsatorische Verschiebung des linken Vorhofes (während der Kammersystole) durch die expandierende Aorta verursacht ist, als, wie man allgemein annimmt, durch Ausdehnung des Vorhofes, die auf einen regurgitierenden Blutstrom von der linken Kammer beruht. Die Bezeichnung »Aorta-diastolische-Verschiebung« wird für dieses Phänomen vorgeschlagen.

RÉSUMÉ

La contradiction entre la conception généralement admise sur la nature de «l'expansion systolique de l'oreillette gauche» et les constatations du chirurgien par la palpation intracardiaque («pas de jet par régurgitation») a poussé à faire une étude radiokymographique de 131 malades atteints de cardiopathie mitrale.

L'auteur en conclut que ce signe est causé par un déplacement pulsatoire de l'oreillette gauche (pendant la systole ventriculaire) dû à l'expansion de l'aorte, plutôt que par une expansion de l'oreillette due à un courant de sang rétrograde venant du ventricule gauche, comme on l'admet généralement. Il propose d'appeler ce phénomène «déplacement aorto-diastolique».

REFERENCES

1. Abelman, W. H., Ellis, L. B. and Harken, D. E.: The diagnosis of mitral regurgitation. Am. J. Med. 15 (1953), 5.
2. Anderson, T.: Electrokymographic studies of left auricular movements. Acta radiol. 38 (1952), 81.
3. Davison, P. H. and Epps, R. G.: The left auricular electrokymogram in mitral stenosis. Brit. Heart J. 16 (1954), 49.
4. Elkin, M., Sosman, M. C., Harken, D. E. and Dexter, L.: Systolic expansion in the left auricular in mitral regurgitation. New England J. Med. 246 (1952), 958.
5. Froment, R., Gonin, A. and Callavardin, L.: L'expansion systolique auriculaire peut-elle être prise comme critérie d'insuffisance mitrale? Arch. d. mal. du cœur 43 (1950), 678.
6. Hirsch, J. S. and Gubner, R.: Application of roentgenkymography to study of normal and abnormal physiology. Am. Heart J. 12 (1936), 413.
7. Lehman, J. S. and Curry, J. L.: A correlation of roentgen and surgical findings in two hundred cases of rheumatic mitral valvular disease. Am. J. Roentgenol. 71 (1954), 599.
8. Lemke, H. H.: Gibt es eine für die Insuffizienz der Segelklappen typische Vorhofspulsation? Fortschr. Röntgenstr. 59 (1939), 372.
9. Logan, A. and Turner, R.: The diagnosis of mitral incompetence accompanying mitral stenosis. Lancet 263 (1952): 2, 593.
10. Logan, A. and Turner, R.: Mitral stenosis. Lancet 264/1 (1953), 1007 and 1057.
11. McKusick, V. A.: The study of mitral regurgitation by roentgen kymograms. Am. J. Roentgenol. 71 (1954), 961.
12. Pauzner, Y. M.: Personal communication.
13. Roesler, H.: Clinical roentgenology of the cardiovascular system. Charles C. Thomas, Springfield-Baltimore 1943.
14. Schlegel, B. and Schoelmerisch, P.: Röntgenkymographische und phonokardiographische Untersuchungen zur Haemodynamik des linken Vorhofs bei Mitralstenosen. Ztschr. Kreislaufforschung 42 (1953), 848.
15. Wood, P.: An appreciation of mitral stenosis. Part II. Investigations and Results. Brit. Med. J. 1954, p. 1113.
16. Zdansky, E.: Röntgendiagnostik des Herzens und der grossen Gefässe. Springer, Wien 1949.
17. Zoob, M.: The esophageal pulse in mitral valve disease. Brit. Heart J. 16 (1954), 39.

FROM THE ROENTGENDIAGNOSTIC DEPARTMENT (DIRECTOR: PROF. KNUT LINDBLOM), KAROLINSKA SJUKHUSET, STOCKHOLM, SWEDEN

A NEW IDENTIFICATION PRINTER FOR ROENTGENOGRAPHIC FILM

by

Ove Mattsson

The marking of roentgen films with the name of the patient and hospital as well as the date of the examination should be simple and easy to perform; the data visible on the film should be distinct so that possibilities of error are excluded. For more than two decades the identification printer developed by Å. ÅKERLUND and described in 1931 has been used. Its principle of construction is the one which, in Sweden, has prevailed almost exclusively. The identifying legends are recorded photographically on the film and are developed together with the roentgenogram. ÅKERLUND's apparatus has satisfied very high demands, both as regards efficiency and simplicity. It has been used in a great number of roentgen departments up to now practically in its original form.

The operation of the ÅKERLUND identification printer is well known. Pertinent data typed on a card of suitable weight and density are recorded on the film by contact printing. Small stencil plates with the name of the hospital and the date are contained in the printer. Depression of a stamping plate holds the film in position, and the printing light is switched on. It should be pointed out that many constructions utilize similar principles. S. R. KJELLBERG, in 1939, described a marking device incorporating a projection system with a lens giving a reduced-scale reproduction of the necessary data on the film. Principles resembling that of ÅKERLUND with contact printing were used in the apparatus described by C. ASHWIN (1941) and W. S. ALTMAN (1946). The preparation of identifying legends on roentgenographic films is now generally done by means of photographic recording with identification printers.

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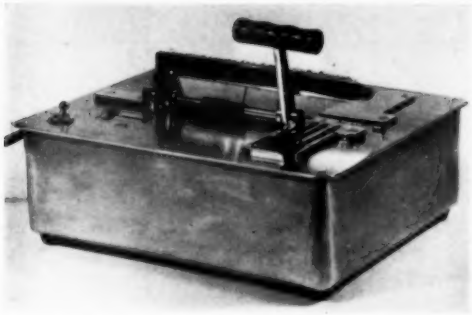

Fig. 1. The printer may be recessed into the table or used as a separate unit. The lever with handle moves the film against the text square.

Various factors have made it desirable and necessary to construct a modernized identification printer. The introduction of new systems for patient registration has been followed by a demand for increased space for identification data, since more information has to be included. The patient's date of birth is of importance, as it is upon this that the registration systems are now based. It is desirable that the area reserved for roentgenographic identification should have a width adapted to the standard size of metal plates used in 'Citograph' or 'Addressograph' systems, which have been introduced in many hospitals to save time and simplify clerical work. On these plates all required data on a patient are assembled and may be reproduced identically in the hospital register, remittances, roentgenographic reports, and so on. There are also roentgen departments which have introduced these systems exclusively for internal use. Ordinarily, only the upper line of the text will be used for the identification of the films. On the other hand, the complete information contained on the plate may be reprinted on reports, film envelopes, etc. In this connection the carbonized snap-out form for identification printers described by A. V. BURDICK (1955) ought to be mentioned. It eliminates duplication of work by carbon paper.

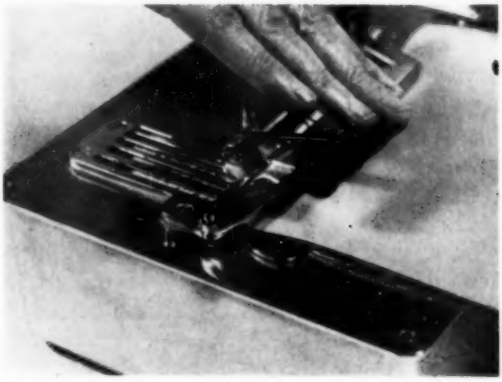

Fig. 2. Printing performed by depressing the lever, the film being flattened against the text square. Correct exposure obtained automatically.

Besides these new demands regarding the data to be reproduced on the film, consideration of certain qualities of the printer should not be overlooked. A uniform exposure in each printing operation is desirable in order to secure optimal visibility of identification. It is necessary that the printing exposure should not depend upon manual operation. This makes

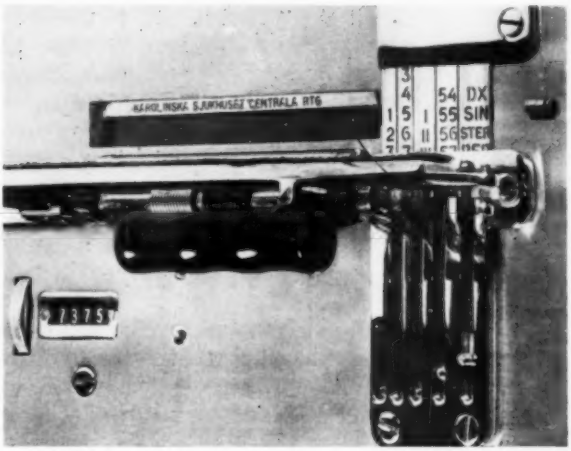

Fig. 3. Printer seen from above. Note system of date-marking by means of small metal strips with cut-out figures.

some sort of timer indispensable. The construction of the apparatus should permit recessing into the table of the darkroom, if necessary. In order to obtain a general idea of the development of the apparatus used for identification, printers from various manufacturers were studied and tested, among them one of Danish, two of English, and one of German construction. None of these, in the author's opinion, showed any important superiority regarding essential details. There appeared, therefore, no reason to change the basic principles of ÅKERLUND's apparatus.

The new printer is shown in Fig. 1. It is made of polished stainless steel; this material gives a neat and agreeable appearance, and is in every respect well suited for the purpose. The upper surface is flat, the box being of a suitable shape for recessing into the darkroom bench so that it lies flush with the latter. This arrangement provides more space and facilitates working. The unit may also be used separately if required.

Different timing systems have been studied and tested. Mercury relays, as used in certain designs, seem to be too sensitive, and are also

Fig. 4. Film marked with printer.

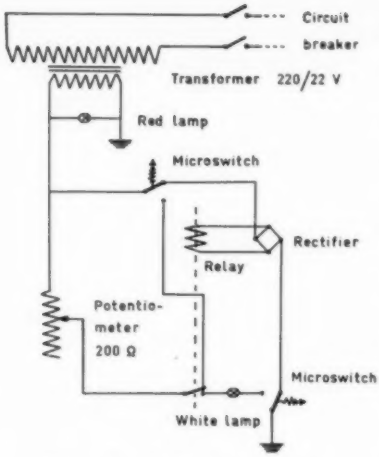

Diagram 1. Wiring diagram.

expensive. The use of a large capacitor, charged to a predetermined potential and discharged during the exposure through a special lamp, seems to be somewhat complicated, but has been used in other printers. Mechanical timers are both simple and reliable, but the simplest of all methods of timing is to use a time-lag relay of the type employed in telephony. This relay gives a short constant impulse of current and is very reliable. If variations from the standard time of exposure are desired, the relay is switched out of circuit and the exposure performed manually. Adjustment of the intensity of the exposure to the particular roentgenographic material is effected by a continuously variable resistor, adjustable by an external screw.

The film is held in position by means of a lever with a handle for easy manipulation. The printing is effected by depression of the press plate so that the film is flattened against the data square (compare Fig. 2). The exposure, of a fraction of a second, is then made automatically; this is indicated by a slight clicking sound and the light from the lamp may be observed through a small hole. For checking the name, etc., a continuous red light illuminates the text square and in order to obtain homogeneous illumination of the data square light conductors of plexiglass are used; the sources of light are placed laterally, which is a great advantage from the point of view of construction.

A new method employing metal strips as stencil plates for dating gives easy positioning. The strips may be pushed into small slides, and the desired dates easily readjusted. In Fig. 3, in which the apparatus is shown from above, the dating arrangement is seen on the right. The name of the hospital is so placed that the space is used more economically than in previous designs.

In order to obtain statistic reports of the work of the roentgen department, a counting arrangement seems to be of a certain value. The principle of such counting has previously been described (MATTSSON 1951), and may be of importance for the correct replenishment of the developer. A counter may be placed inside the housing of the apparatus, and its face is then visible on the upper surface of the box as to the left in Fig. 3. Each identification operation produces an impulse and a progression of the counter.

The appearance of the identification data in the film is shown in Fig. 4. The wiring of the electric circuits is given in Diagram 1. It should be pointed out that the bulbs used in the apparatus are of standard type, and run at below normal voltage with consequent prolonged life.

The prototype of the identification printer, which has been developed in cooperation with K. FENZ of G. Schönander AB, has been in use in Roentgen Diagnostic Department B of Karolinska Sjukhuset for about a year and has marked about 75,000 films practically without maintenance.

SUMMARY

A new identification printer utilizing the well-known principle developed by ÅKERLUND is described. The printer satisfies the latest demands of the registration systems employed in hospitals.

ZUSAMMENFASSUNG

Ein neuer Filmmerkungsapparat wird beschrieben, bei dem das von ÅKERLUND entwickelte und wohlbekannte Prinzip benutzt wird. Der Apparat befriedigt die neuesten Forderungen der in Krankenhäusern benutzten Registrierungssysteme.

RÉSUMÉ

Description d'un nouveau marqueur de films utilisant le principe bien connu mis au point par ÅKERLUND. Ce marqueur répond aux exigences les plus modernes des systèmes de classement en usage dans les hôpitaux.

REFERENCES

ÅKERLUND, Å.: Ein Lichtstempel für Filmbeschriftungszwecke. Acta radiol. 12 (1931), 95.

ALTMAN, W. S.: Custom made film identification marker. Am. J. Roentgenol. 55 (1946), 664.

ASHWIN, C.: Actinic film-marking. Radiography 7 (1941), 6.

BURDICK, A.: Carbonized snap-out form for identification printers. Med. Radiogr. and Photogr. 31 (1955), 80.

KJELLBERG, S. R.: Ein neuer Typus eines Filmmerkungsapparates. Acta radiol. 20 (1939), 253.

MATTSSON, O.: Automatic counting of radiographs — an aid in processing control. Acta radiol. 38 (1952), 215.

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A NEW TELEGAMMA APPARATUS

by

Bo Lindell and *Rune Walstam*

Radioactive cobalt can now be easily obtained making it possible to construct therapy apparatus having several essential advantages over the teleradium units previously used. Other radioactive isotopes, notably Cesium 137, may be used for the same purpose.

Teleradium treatment has been employed principally in England and Sweden (5, 8, 15, 17, 18), in the treatment of tumours which lie from one to five or six centimeters below the skin surface. Treatment is restricted to tumours in this depth range by the scarcity of radium, which necessitates working with comparatively low activities, at the most a few grams. Bone is often present at these depths, especially in the oral and nasal cavities. In bony tissue gamma rays give a more homogeneous distribution than roentgen rays and teleradium has therefore been the treatment of choice in precisely the range of depths in which the shortage of radium has limited its application.

By the early twenties teleradium units had been constructed which gave optimum depth doses with the quantities of radium then available. In 1924 STENSTRÖM (22) described an arrangement which produced a conically convergent beam; it contained several small sources which were arranged in a circle behind a conical diaphragm. Ever since then teleradium units have usually consisted of several small radium tubes distributed over a large area. This design has been chosen partly because of the low specific activity (a few curies per cc) and high self-absorption of radium, and partly to increase the relative depth dose.

Since 1951 several authors have described the conversion of teleradium units for use with radioactive cobalt. There has been a tendency to use relatively high activities, not only to shorten the treatment time, but

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also to increase the treatment distance (10, 13, 14, 16, 21, 24). When the old extended-source principle is used the fields become rather diffuse and a penumbral effect is obtained, giving almost the same spread at the edge of the beam as that produced by the scattered radiation in a roentgen ray beam. In cases where a high specific activity is not essential it therefore appears to be most convenient to use cesium (where, however, specific activities equivalent to 40 grams of radium per cc might be obtained), since the units may then be built with better protection while retaining their comparatively small dimensions.

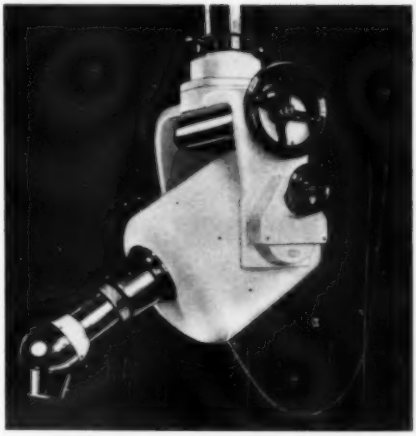


Fig. 1. Overall view of apparatus, showing lead protective container suspended from ceiling and treatment head into which the cobalt cylinder is fed before treatment.

In constructing the unit now described, we have not attempted to widen the scope of the teleradium technique as developed at Radiumhemmet to include greater treatment distances, but have endeavoured on the other hand to design a unit which allows some refinement of the old technique together with shorter treatment times and a greater variety of set-ups.

The general design is the same as in the teleradium units described by SIEVERT (20) and BENNER (1), *i. e.* the apparatus consists of a protecting lead container and a treatment head into which the source can be fed automatically (Fig. 1). The apparatus is suspended from the ceiling and has the same range of movement as the teleradium unit; it is heavy but this is a disadvantage only to the manufacturer and not to the user, the apparatus being neither bulky nor cumbersome. The favourite method of source transfer in Great Britain since 1935 has been the use of air pressure to move the source into the treatment head from a storage safe situated some distance from the unit (11). Since the positioning and direction of the beam depend essentially on the design of the treatment head and not on the location of the storage safe, we have employed Sievert's principle which, in our opinion, is the more reliable.

The shape of the treatment head in this construction is unusual. In place of an extended source we employ only one small cylinder (6 mm in diameter). This gives a depth dose along the field axis which is somewhat inferior to that of other units. When multiple field techniques are used, however, we can obtain higher depth doses than are possible with

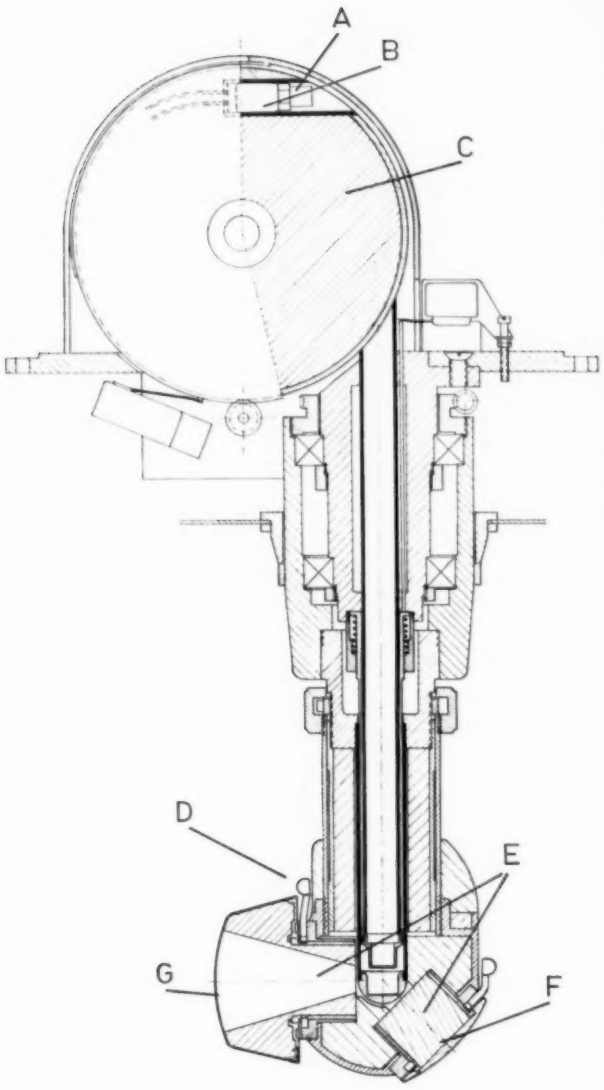


Fig. 2. Treatment mechanism: A — source container; B — guiding plug; C — lead drum and revolving disc; D — treatment head; E — beam apertures; F — lead plug; G — beam nozzle.

an extended source unit (cf. Fig. 6). Since the source is for all practical purposes a point, we are, in addition, able to use diaphragms for defining different fields without reducing the intensity. The sharply defined fields make it possible to protect sensitive organs (*e. g.* the patient's eyes). Though we have constructed the apparatus for radioactive cobalt, it may, if desired, be loaded with cesium; cobalt can be produced, however, with a higher specific activity.

The mechanism which transports the source into the treatment head is shown in Fig. 2. The basic idea is the same as in Benner's design but, since the moving parts are very small, the final construction has been modified. The source is hermetically sealed in an aluminium capsule which has a hexagonal head and carries a threaded pin; this can be fastened to a cylindrical guiding plug which can, by means of a spiral wire, be pushed out of the storage container and down through the tube supporting the treatment head. The wire is fed in an axial direction (producing minimum friction) from a

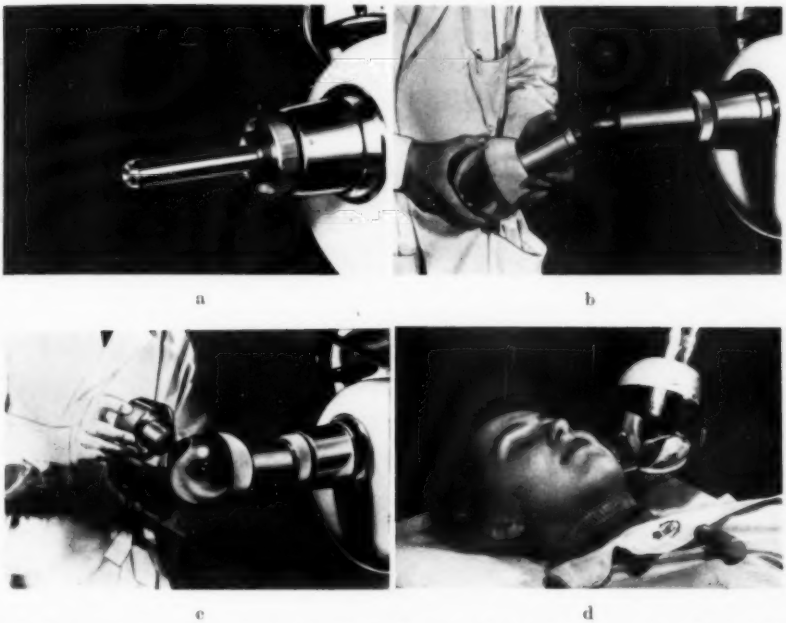
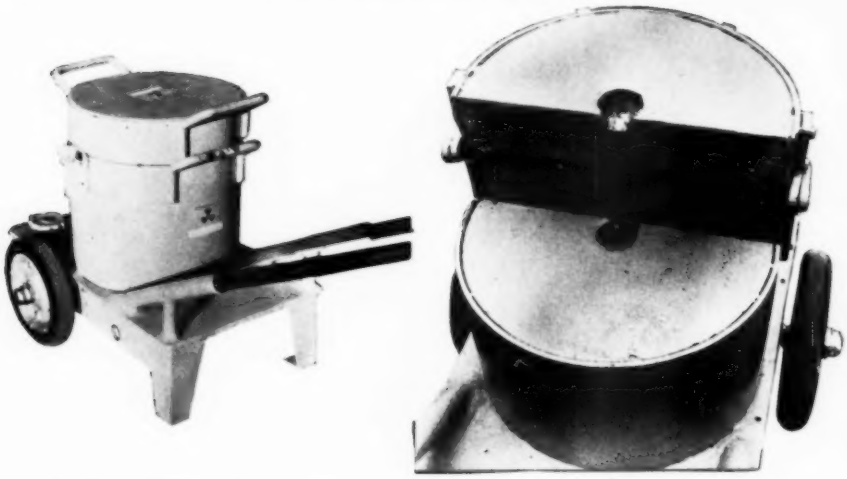


Fig. 3. Construction of treatment head. a. Bare steel pipe. b. Mounting the treatment head. Steel pipe covered with a lead cylinder. c. Beam nozzle being fitted into one of the two apertures. d. Treatment head applied to patient in frequently used set-up.

groove in a revolving disc. The disc also carries a lead drum containing a hole for the guiding plug and the aluminium capsule. When the source is in the 'safe' position in the centre of the container, there are at least 12 centimeters of lead shielding in all directions. The feeding disc and the lead drum can be turned as a unit through 90° into a position in which the drum is locked, the hole carrying the source being fixed at the bottom of the storage container and connected with the treatment head support. The feeding disc then turns through an additional revolution and pushes out the wire, which then feeds the source through the tube into the treatment head. The closed position of the source is well defined by means of a mechanical stop. The feeding mechanism was tested by subjecting it to 80,000 up-and-down movements before its design was approved.

In teleradium units the pipe connecting the container and the treatment head is usually curved because the radiation can be used only in a direction perpendicular to the plate on which the sources are mounted. In our case, however, the source is practically point-shaped and it is



a b

Fig. 4. Auxiliary equipment for carrying source and loading unit. a. Lead cart with covers in position. b. Cart opened to show hollow key and source capsule.

possible (at least theoretically) to make use of the radiation in almost any direction. The steel pipe connecting the container and the treatment head has therefore been straightened and the treatment head, which can be turned about its shaft, provided with two apertures. The opening which is not in use is closed by a lead plug while the other one is fitted with a convenient beam nozzle. All these arrangements combine to make the application of the beam to the patient easy and also permit a very close approach even if the available space is limited.

Fig. 3 a shows the bare steel pipe through which the guiding plug and the source container are fed. It ends in an aluminium or lucite hemisphere which can easily be unscrewed when the source has to be replaced. The pipe diameter is 30 mm, making it possible to bring the source close to the object irradiated. In a few cases the steel pipe was inserted into the mouth of patients with intra-oral tumours. This has been done when it was urgently necessary to give additional doses after conventional treatment.

In order to reduce the radiation transmitted from the source during its journey between the safe and the treatment head, the steel pipe is shielded by a lead cylinder. Fig. 3 b also shows how the rotating head with its two apertures can be easily mounted. Fig. 3 c demonstrates how a beam nozzle is fitted into the opening to be used. Finally Fig. 3 d shows the complete treatment head applied to a patient in a frequently used set-up.

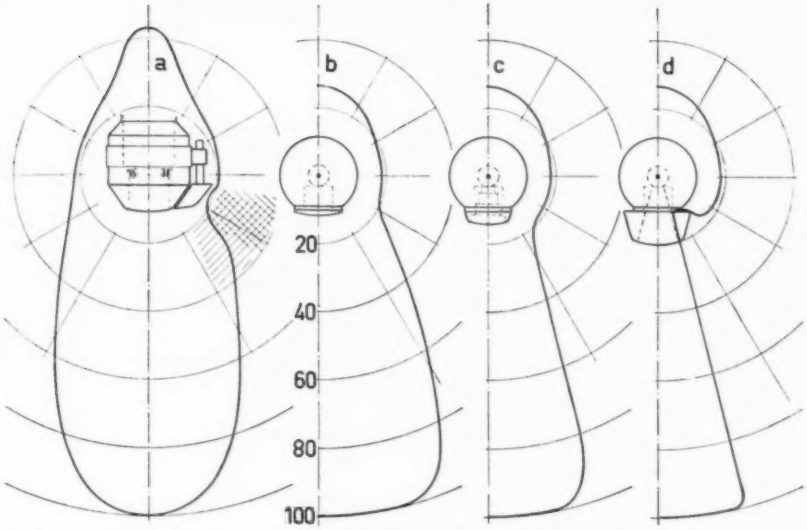


Fig. 5. Transmission through treatment head. a. Radiumhemmet 5 g teleradium unit. b. Cobalt, 5.8 cm S. S. D. circular field, no additional beam definition. c. Cobalt, 7 cm S. S. D. circular field, diffuse edge. d. Cobalt, 10 cm S. S. D. circular field, sharp edge.

This apparatus has been in use for treatment at Radiumhemmet since December 1954. It was then loaded with 8.5 curies of radioactive cobalt in the form of a cylinder 6 mm long and 6 mm in diameter, produced at Harwell.

Loading and reloading of the source may be easily carried out, with the help of a special lead cart which reduces the irradiation (Fig. 4). The cart may also be used as a transport container, which is loaded by the provider of the source. In the centre of the cart there is a remotely-controlled hexagonal key which fits the head of the guiding plug. In loading, the treatment head is unscrewed and the cart is then placed directly under the apparatus. The operator takes shelter behind the cover of the lead container on the cart, from which protected position he lowers the guiding plug towards the hollow key holding the source capsule. He then fastens the latter by remote control. It is possible to provide the cart with two hollow keys for the old and new sources, respectively.

The small source dimensions have made it possible to give the main protective container of the apparatus an effective shape. The total weight of the suspended mass is 350 kg and 9 curies in the 'safe' position give less than 7 mr per hour at one meter from the source. During the first protection check it was discovered that there was a certain risk of sharply-

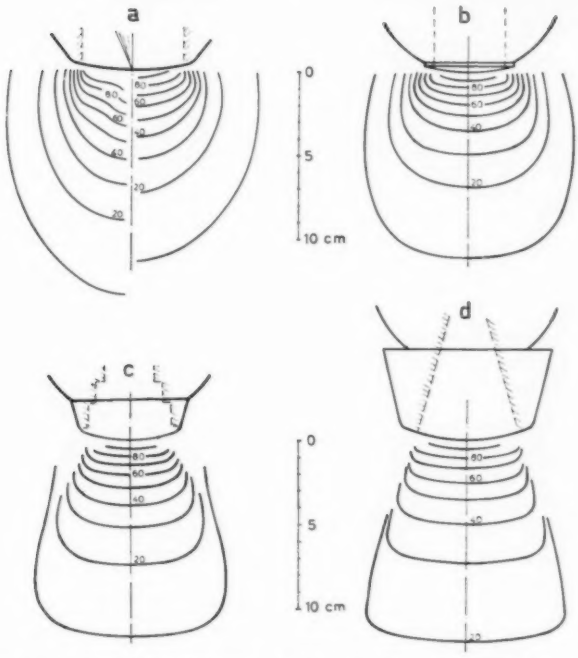


Fig. 6. Isodose curves with different beam nozzles. a. Radiumhemmet 5 g teleradium unit. b. Cobalt, 5.8 cm S. S. D. circular field, no additional beam definition. c. Cobalt, 7 cm S. S. D. circular field, diffuse edge. d. Cobalt, 10 cm S. S. D. circular field, sharp edge.

defined local leaks. Since these could not be adequately guarded against during construction, they were eliminated with the aid of tungsten plugs after the source was installed.

Because of the small source size the treatment head may also be given an effective shape. The patient's total body dose is about the same as in teleradium treatment. At the short source—skin distances used in telegamma therapy it is not, however, possible to specify the protection by giving the dose outside the field as a percentage of the dose at the centre, as is usual in deep therapy. The inverse square law alone decreases the dose-rate at 60 cm to one per cent of its value at 6 cm. Hence, even without a protective shield the patient's total body dose is quite small. How absorption in the treatment head itself affects the dose outside the field is shown in Fig. 5.

The treatment began with three different beam nozzles defining circular fields from 5 to 6 cm diameter, and using source—skin distances of

5.8, 7, and 10 cm respectively. The isodose curves for these nozzles are shown in Fig. 6. The dose distribution in a wax phantom was measured with a Bomke dose-meter, calibrated against known radium tubes. These measurements were completed by making a dose distribution survey with Eastman Kodak 548-0 film which is very insensitive and has a very thin emulsion which does not disturb the tissue equivalence of the phantom. For direct dose measurements at the edge of the beam and near the surface of the phantom we used very small 'needle chambers' (constructed by BENNER) with a sensitive volume of only 2 cubic millimeters. (The Bomke dosemeter was placed at our disposal by courtesy of the Rotation Laboratory staff.)

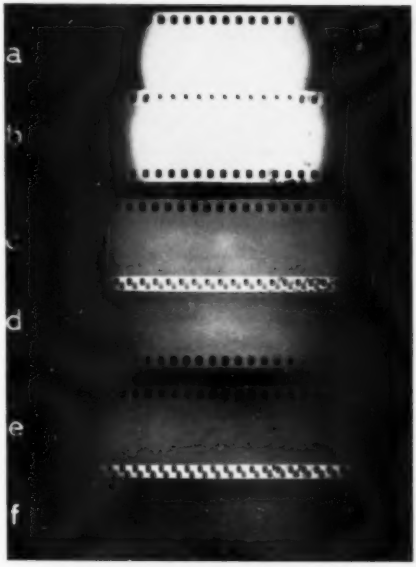


Fig. 7. Depth dose distribution illustrated by means of film phantom. a, c, e: Cobalt unit with 10 cm S. S. D. beam nozzle. b, d, f: 5 g radium unit, without central cone. Films a and b placed at depth of 0.02 cm, c and d at 3.7 cm, e and f at 5.7 cm.

The films shown in Fig. 7 were placed at different distances under the treatment head perpendicular to the beam. They illustrate the difference in dose distribution between the five gram radium unit and the cobalt unit, the latter giving a more homogeneous dose inside the treatment beam, a lower dose outside the beam, and a sharp edge *if desired*. A sharp edge is preferable in most cases, except at the boundary between two adjacent fields where there is a risk either of overlapping or of leaving a gap between the beams. In all other cases it must be remembered that the dose outside the isodose contour representing the threshold value for tumour cell destruction may very well be reduced to zero. It falls upon the physician to say what tissue should fall inside the threshold contour and the level to which the dose within it should amount. If he cannot, a diffuse treatment beam will not make the result better, if he can, it will certainly make it worse.

Of our first series of beam nozzles only the one for a 10 cm source—skin distance had a diaphragm which produced a sharp edge. The others were made to give a dose distribution as close to that of the radium unit as possible. It was then possible to treat the patients according to the old routine until new methods had been developed.

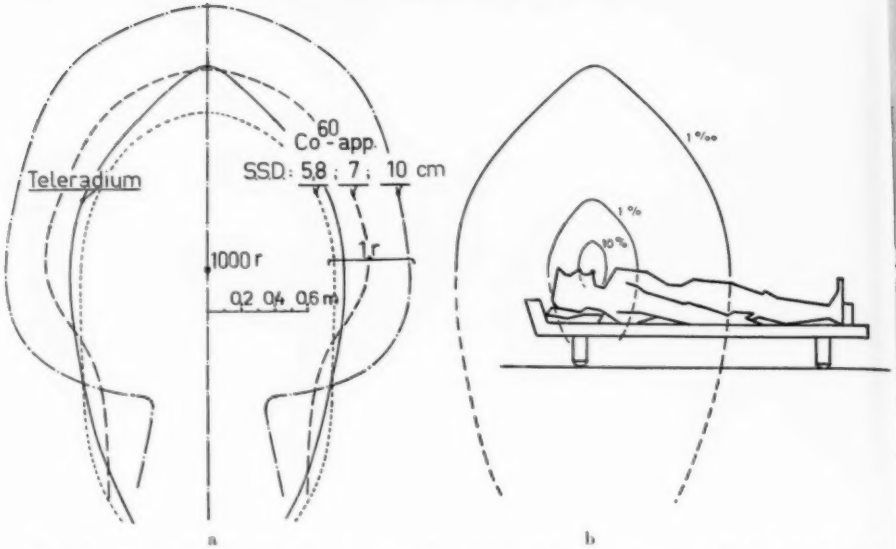


Fig. 8. Dose distribution in air around treatment head, as measured with cardboard ionization chamber. a. Different beam nozzles compared. All curves refer to 1,000 r extrapolated surface dose. b. Air-dose distribution in teleradium treatment, in relation to size of patient.

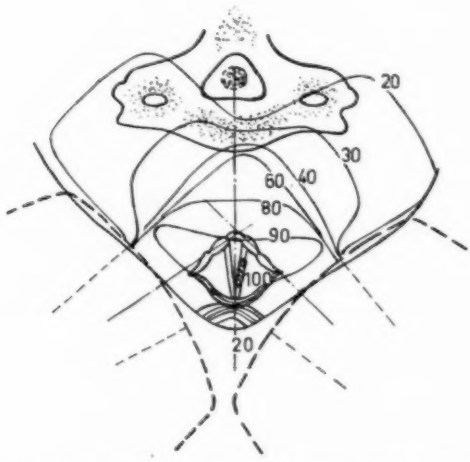
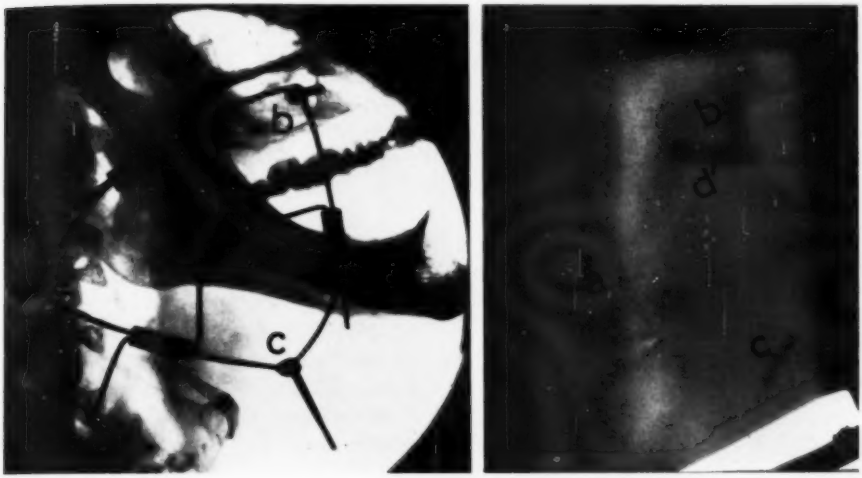


Fig. 9. Isodose curves for a planned two-field treatment of carcinoma of the larynx, using a 10 cm S. S. D. beam nozzle with 3 × 3 cm square fields.



a b

Fig. 10. Photographic control of treatment of carcinoma of right side of tongue. a. Roentgenogram of patient in treatment position. Centres of the three right-hand fields (a, b and c) indicated by wire jig. b. Photographic control (using the cobalt as radiation source) of additional 3.5 × 6 cm rectangular field, applied from left side with 10 cm S. S. D. beam nozzle. Fields a, b, and c: lead indicators at centres of right-hand fields, d: tungsten indicator at primary tumour. Note sharp exit contours of beam.

The unit gave the following output when the source activity was 8.5 curies:

5.8	cm	S. S. D.	— 55	r/min
7	»	»	— 34	»
10	»	»	— 20	»

The product of dose-rate and source—skin distance squared was unequal for the three different nozzles, possibly as a result of the varying amounts of radiation scattered from the walls of the diaphragm. The treatment times were of the order of 10 to 50 minutes, and hence 30 curies may be considered an optimum activity for these source—skin distances. Higher activities would need less than three minutes at the shortest distance and would be difficult to handle safely.

The absorption of radiation in the treatment head (shown in Fig. 5) is such as to reduce the patient's total body dose to a level determined by the treatment time with each nozzle. It is seen from Fig. 8 that this level is about the same as in teleradium treatment though it may at first sight seem unexpectedly high. It must, however, be remembered that the true dose distribution in a patient irradiated with

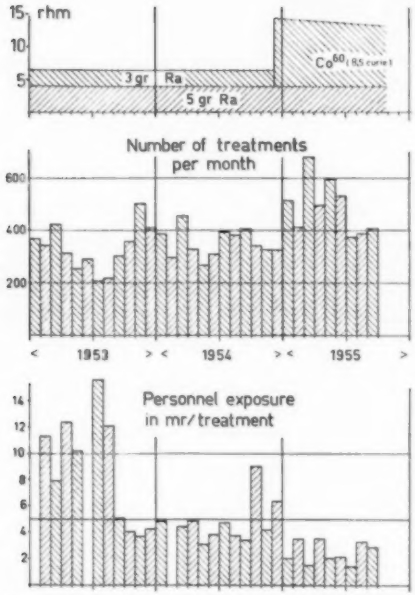


Diagram 1. Amount of radioactive substances, number of treatments per month and personnel exposure in Teleradium Department. Dose given in mr/treatment in order to eliminate effect of variations in numbers of patients and personnel. Radiation protection work was intensified in 1953 and the cobalt replaced 3 g of Ra in December 1954.

roentgen rays gives a body dose of the same order, because of scattered radiation. Since in practice we have found no indications that a decrease in total body dose was necessary, we are content to reproduce teleradium conditions in the present apparatus.

As the source is comparatively cheap it pays to exchange the cobalt at least every second year. The dose-rate and dose distribution must, however, be carefully checked each time the cobalt is changed. Allowance is made for the decay of the cobalt by revising the treatment times every three months.

The unit has not been running long enough to justify a discussion of the clinical results or even of the best treatment techniques. Nevertheless, we have made a number of new beam nozzles which produce rectangular and triangular fields, all with sharp edges. Experimental rectangular fields, 3×6 cm and 3×3 cm in size were also used in the early stages and proved useful. The small source size preserves the field shape at a depth, a result impossible with the source diameters used by those workers (7, 13) who attempted to cover a large area with non-circular fields. Fig. 9 shows a planned two-field treatment; the actual dose distribution in the patient is somewhat more diffuse because the setting-up introduces some error. The planning of multiple-field treatments is usually carried out by means of the wire jig method (Walstam 23).

Before treatment it is possible to make a photographic check of the accuracy of beam direction (Fig. 10). The film will show very little contrast but may still be interpreted if air-filled cavities are present, or if lead or tungsten pellets are placed on the skin. It is reassuring to note that the lack of contrast indicates a homogeneous dose.

The unit may of course also be used for other radiographic purposes, for example to examine roentgen ray tubes in which it is suspected that

the tungsten target has worked loose from the copper body of the anode (BENNER, 3, 4).

The operation of the unit is worthy of note. The protection of personnel has always been a problem in teleradium work (1, 2, 9, 12, 19, 20). We believe that the new unit permits safer handling than was previously possible. There is adequate protection during the setting-up of the patient, and during treatment the operator is completely shielded in the control room. Previously the operator had received weekly doses of up to 1 r if, without first returning the radium to the 'safe' position, she entered the treatment room to attend to the patient. We have now installed an alarm which rings if the door of the treatment room is opened while the source is not in the 'safe' position. The need for entering a room, is in any case now much reduced. Benner's positioning indicator (2) which produces a signal in the control room as soon as the patient loses contact with the beam nozzle, has been built into the unit; the patient hears the signal and adjusts his position without the help of the operator.

All parts of the electric circuit which need to be serviced are placed in the control panel outside the treatment room. Most of the movable parts may be reached while the cobalt is in the 'safe' position. It is possible to free the mechanism from the heavy lead container as a complete unit once the source has been taken away. In case of a power failure the source may be moved to the 'safe' position with an emergency handle.

SUMMARY

A new telegamma unit, loaded with 8.5 curies of radio-active cobalt and used for treatment at Radiumhemmet since the end of 1954, is described. The unit is intended for treatment with a source—skin distance of 6 to 10 cm. The employment of a 6 mm source makes it possible to use diaphragms defining different field sizes and shapes, well preserved even at a depth. Apart from other advantages, it is believed that the new unit gives safer handling than previous constructions.

ZUSAMMENFASSUNG

Ein neuer, mit 8.5 Curie radioaktivem Kobalt geladener Telegammaapparat, der seit Ende 1954 im Radiumhemmet in Betrieb ist, wird beschrieben. Der Apparat ist für Behandlungen mit 6—10 cm Präparat—Hautabstand konstruiert worden. Durch Verwendung verschiedener Behandlungstubusse können Strahlenbündel verschiedener Form ausgeblendet werden, die auch in der Tiefe scharf abgegrenzt sind. Abgesehen von anderen Vorteilen scheint die Handhabung des neuen Apparates bedeutend sicherer als die früherer Konstruktionen zu sein.

RÉSUMÉ

Les auteurs décrivent un nouvel appareil de télégammathérapie, chargé avec 8.5 curies de Cobalt radioactif, qui est en fonctionnement au Radiumhemmet depuis la fin de 1954. Cet appareil a été construit pour des traitements avec une distance source—

peau de 6 à 10 cm. L'emploi d'une source de 6 mm permet d'utiliser des diaphragmes délimitant des champs de dimensions et de formes variées, qui restent bien définis même à une profondeur de quelques cm. Outre d'autres avantages, il semble que le maniement de cet appareil soit plus sûr que celui d'appareils antérieurs.

REFERENCES

1. Benner, S.: A new teleradium apparatus. Acta radiol. 28 (1947),765.
2. — On the use of radioactive substances in Sweden, with special reference to irradiation risks and protection measures. Acta radiol. 33 (1950), 281.
3. — Fehler in Röntgenröhren, die mit Hilfe von Gammastrahlen-Aufnahmen, nachgewiesen werden konnten. Acta radiol. 25 (1944), 414.
4. — Personal communication.
5. Berven, E.: Development of technique and results of treatment of tumours of the oral and nasal cavities. Am. J. Roentgenol. 28 (1932), 332.
6. Bomke, H.: Das neue Radium-Momentan-Dosimeter nebst principiellen Erörterungen zur Radiumdosimetrie. Strahlentherapie 86 (1952), 345.
7. Burnham, C. F. and Ward, G. E.: Recent developments in protective methods and appliances as used in radium therapy. Am. J. Roentgenol. 10 (1923), 625.
8. Cade, S.: Malignant disease and its treatment by radium. John Wright & Sons Ltd, Bristol 1940.
9. Failla, G.: Design of well-protected radium "pack". Am. J. Roentgenol. 20 (1928), 128.
10. Fry, W. H., Miller, H. and Orton, K. F.: A 50 curie Cobalt 60 teletherapy unit. Brit. J. Radiol. 28 (1955), 8.
11. Grimmet, L. G.: A five-gramme radium unit with pneumatic transference of radium. Brit. J. Radiol. 10 (1937), 105.
12. Kutzin, H. and Klesse, P.: Die Anlage für Teleradiumtherapie mit 10 g Radium in den Kölner Universitätskliniken. Strahlentherapie 93 (1954), 389.
13. Lederman, M. and Greatorex, C. A.: A Cobalt 60 telecurie unit. Brit. J. Radiol. 26 (1953), 525.
14. Lidén, K.: A 10-curie Co^{60} telegamma unit. Acta radiol. 38 (1952), 139.
15. Lysholm, E.: Apparatus for the production of a narrow beam of rays in treatment by radium at a distance. Acta radiol. 2 (1923), 516.
16. Merewether, H., Osborn, S. B. and Wyard, S. I.: The installation of Co^{60} in a gamma ray beam unit. Acta radiol. 37 (1952), 459.
17. Paterson, R.: The treatment of malignant disease by radium and X-rays. Edward Arnold & Co, London 1948.
18. Sievert, R. M.: The new apparatus for teleradium treatment used at Radiumhemmet. Acta radiol. 14 (1933), 197.
19. — On protection against radiation in teleradium treatment. Ibid. 14 (1933), 597.
20. — Two arrangements for reducing irradiation dangers in teleradium treatment. Ibid. 18 (1937), 157.
21. Spiers, F. W. and Morrisson, Margaret T.: A Cobalt 60 beam unit with a source—skin distance of 20 cm. Brit. J. Radiol. 28 (1955), 2.
22. Stenström, W.: Methods of improving the external application of radium for deep therapy. Am. J. Roentgenol. 11 (1924), 176.
23. Walstam, R.: A method for three-dimensional dose-finding in teleradium therapy. Acta radiol. 43 (1955), 477.
24. Editorial, Brit. J. Radiol. 28 (1955), 1.

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MEASUREMENTS ON RADIATION PROTECTION REQUIRED IN THE WALLS OF ROENTGEN DIAGNOSTIC ROOMS

by

Lars-Eric Larsson

In Sweden as in many other countries there are rules for the protective barriers required against undesired exposure to roentgen rays. The necessary protection depends upon the size of the permissible dose which the personnel may receive continuously without suffering any damage to the blood or reproductive organs. In the latest recommendations of The International Commission on Radiological Protection (1), the following permissible weekly doses in millirem are given for whole-body exposure to ionizing radiation: skin 600, blood-forming organs 300. These requirements are assumed to be met when the dose measured in free air does not exceed 300 mr/week.

From the tabulated values in the recommendations of the ICRP, the protective barriers required can be calculated, *e. g.* for roentgen diagnostic rooms. For the primary radiation beam at a distance of 2 m from the tube target, and with a working extent of 100,000 milliampere-seconds (mAs) per week, a lead thickness of 1.7 mm at 100 kV and 2.3 mm at 150 kV is required at direct current potential.

In Sweden, roentgen-ray workers are prevented as far as possible, from receiving a weekly dose exceeding 100 mr (2). Consequently, the practical lead thicknesses required for diagnostic rooms are 2 mm at 100 kV and 3 mm at 150 kV under the same conditions as above.

These required lead thicknesses seem to be the result of theoretical calculations, in which consideration has been paid to the most unfavourable circumstances, *e. g.* that there is no patient in the beam, and that in

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the course of a week every part of the walls, floor, and ceiling of the roentgen rooms is struck by the amount of radiation generated from 100,000 mAs.

The investigation was performed in the Roentgendiagnostic Department of Karolinska Sjukhuset, in order to ascertain whether the calculations mentioned above correspond to reality.

Measurements. Ionization chambers were put up on the walls of roentgen diagnostic rooms used for various purposes, in order to measure the amount of radiation which, in practice, strikes the protective barriers. The measurements were made with double ionization chambers, described earlier by SIEVERT and WALSTAM (3). These chambers have an extensive range of measurement, *i. e.*, from 10 mr up to 2,000 mr, and are sufficiently independent of energy for this kind of measurement. They were fixed to the walls with special adhesive plaster containing only low atomic materials, and were placed between 1.0 and 1.5 m above the floor. The chambers were read once a week. Because of a shortage of chambers, it was not possible to set up others during the time necessary for reading. Consequently, a correction factor was needed for these periods. This factor was calculated to be 10 per cent of the measured dose value and was added to this value.

The measuring points were not chosen on any rigorous principles, but due consideration was paid to certain factors. The chambers were placed at those points on the walls on which the primary beam impinges and where — with regard to the placing of the examination stands — the greatest amount of scattered radiation may be expected. Measurements were made on all the walls of a room.

The investigation was made in

1. Rooms for combined fluoroscopy and roentgenography, *e. g.*, for examination of the lungs and alimentary tract.
2. Rooms for roentgenography of the skeleton.
3. Rooms for urologic examinations.

It was impossible to obtain a specification of the number of milliampere-seconds used in the rooms during the measurements. The Roentgendiagnostic Department of Karolinska Sjukhuset is, however, one of the largest in Sweden, and its rooms do not seem to allow an increase in the number of examinations. The investigation was therefore performed under maximum working conditions.

In the course of the measurements, the physicians and nurses moved between the rooms, according to the working schedule of the department. This eliminated the risk of the results being dependent on a personal examination technique.

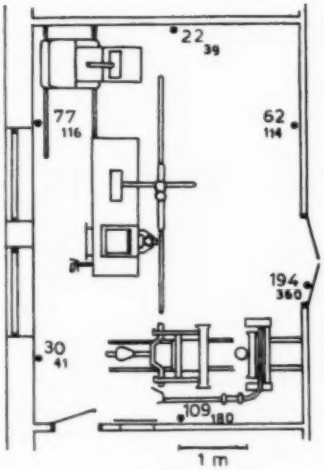

Fig. 1

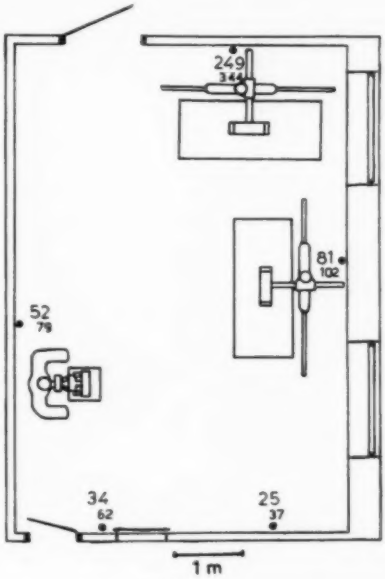

Fig. 2

Fig. 1. Room with fluoroscopic stand, universal roentgen table, and biplane film-changer for examination of lungs and alimentary tract. 4-valve apparatus up to 150 kV.

Fig. 2. Room with two Potter-Bucky tables and skull table for examination of skeleton. 4-valve apparatus up to 100 kV.

The large figures denote mean doses in mr/week and the small figures highest measured doses in mr/week.

Altogether 408 double measurements were made in 9 roentgen diagnostic rooms. The measurements took place:

in	5	rooms	during	10 weeks
»	1	room	»	7 »
»	3	rooms	»	5 »

The apparatuses in the laboratories were of 4-valve or 6-valve type and the total filter of the tubes was 1 to 2 mm aluminium. Only 15 dose values were rejected. The reasons for rejection were dropped chambers or apparent faults in the chambers.

Results. The distribution of the dose values recorded is shown in Table 1. Only 9 weekly doses exceeded 300 mr and none was above 400 mr.

Table 1

Type of Room	F = fluoroscopy R = roentgenography	Number of dose values	Weekly dose in mr 0–49	50–99	100–149	150–199	200–249	250–299	300–349	350–399
Lungs and alimentary tract	F + R	176	76	51	33	9	4	1	0	2
Skeleton	R	135	66	42	7	3	8	7	2	0
Urology	R	82	11	26	18	10	5	7	2	3
	Total	393	153	119	58	22	17	15	4	5

Figs. 1, 2, and 3 show the measuring points and the radiation doses to the walls in three roentgen diagnostic rooms. The large figures indicate the mean dose per week and the small figures the highest weekly dose during the measurements.

A statistical analysis of all the dose values from the nine rooms showed that, even with the probability $p = 0.01$, the largest weekly dose did not exceed 500 mr at any measuring point. In other words, during a period of two years there is no statistical chance of this degree of reaching a weekly dose of 500 mr at any measuring point.

It seems to be reasonable to allow 10 mr/week to pass through the protective barriers. This is one-thirtieth of the international recommended permissible weekly dose and one-tenth of the Swedish. On account of the great number of exposures in a diagnostic room during a week, it is extremely unlikely that the personnel will be exposed to an excessive dose-rate if the barriers transmit only 10 mr/week.

For the rooms in question, it appears from the measurements that the following protective barriers in the walls would suffice if 10 mr/week were allowed to pass:

up to 100 kV	0.5 mm lead
» » 150 kV	1 » »

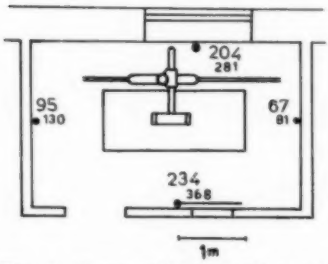

Fig. 3. Room with urologic couch for urologic examinations. 4-valve apparatus up to 100 kV.
The large figures denote mean doses in mr/week and the small figures highest measured doses in mr/week.

These figures were calculated from transmission curves for lead at direct current potential. If the measured wall-doses had been found to be double, this would have implied an increase in the protective lead thickness by one half-value layer; this amounts to only 0.2 to 0.3 mm lead.

In a preliminary report, SMITH and KENNEDY (4) gave an account of corresponding measurements in the USA. Their report contained no further particulars concerning their investigation, and no direct comparison is therefore possible. Their dose values from hospital diagnos-

tic departments seem, however, to be of the same order of magnitude as those found in the present investigation.

Discussion

The measurements show that, for the diagnostic rooms in question, it would have been sufficient to use very small thicknesses of lead (0.5 mm up to 100 kV and 1 mm up to 150 kV) in the protective barriers as compared to those officially recommended. The difference between these figures obtained by measurements on the walls in the actual course of the work and those officially recommended can be explained on the following grounds.

1. To a great extent, only scattered radiation strikes the walls.
2. In rooms with both fluoroscopy and roentgenography, the largest number of milliampere-seconds are consumed in fluoroscopy, when the fluorescent screen absorbs the primary radiation.
3. The patient absorbs most of the useful beam that is directed towards the walls. In Sweden it is not uncommon, particularly in examinations of the stomach with the patient upright, for the fluorescent screen to be drawn down over the cassette.
4. The total quantity of the generated radiation does not strike all parts of the walls. The examinations are distributed over different stands and/or in different radiation directions. In the case of one stand and one radiation direction only, one of the walls receives the maximal radiation.

The rooms for which the measurements are recorded in Figs. 1, 2, and 3 are representative, at least in Sweden, of corresponding roentgen diagnostic rooms. Consequently, the measured dose values could form a basis for a discussion of whether a diminution of the protective barriers in the walls of roentgen diagnostic rooms is possible. This would be of importance, in view of the economic advantages which a diminution of the protective barriers would imply.

The present investigation did not include the floor and ceiling. Owing to considerations of strength, their dimensions often exceed those necessary for radiation protection.

Finally, it must be emphasized that a decrease in the protection in the walls must not lead to a general disregard of radiation protection, since roentgen diagnostic work involves radiation risks for the personnel, which risks are inherent in the roentgen work itself.

Acknowledgement

The author is indebted to Professor KNUT LINDBLOM, Director of The Roentgen-diagnostic Department, Karolinska Sjukhuset, and to his staff for all the help that has facilitated the measurements.

SUMMARY

In order to ascertain the amount of radiation which actually strikes protective barriers, measurements with ionization chambers have been made on the walls of roentgen diagnostic rooms. In no case did the dose values exceed 400 mr/week. A statistical analysis shows that a weekly dose of 500 mr is not reached once in two years under maximum working conditions in Sweden. Some explanations are put forward of the difference between the dimensions of protective barriers given as a consequence of these measurements and those internationally required.

ZUSAMMENFASSUNG

Um die Strahlenmenge, die in der Praxis die Schutzwände der Röntgendiagnostikräume trifft, zu bestimmen, wurden an den Wänden Messungen mit Jonisationskammern ausgeführt. In keinem der Fälle überstieg die Dosis 400 mr/Woche. Eine statistische Analyse zeigt, dass 500 mr in einer Woche während eines Zeitraumes von zwei Jahren nicht ein einziges Mal bei den in Schweden üblichen maximalen Arbeitsbedingungen erreicht wurde. Einige Erklärungen über den Unterschied den Dimensionen der Schutzwände auf Grund der hier angeführten Messungen und den international geforderten Dimensionen werden gegeben.

RÉSUMÉ

Pour déterminer la quantité de radiation qui atteint réellement les revêtements protecteurs, l'auteur a fait des mesures avec des chambres d'ionisation sur les murs de salles de radiodiagnostic. Dans aucun cas les doses n'ont dépassé 400 mr/semaine. Une analyse statistique montre que la dose de 500 mr/semaine n'est pas atteinte une fois en deux ans dans les conditions de fonctionnement maximum en Suède. L'auteur propose quelques explications de la différence entre l'épaisseur des revêtements protecteurs indiquée comme résultat de ces mesures et celle qu'imposent les réglements internationaux.

REFERENCES

1. RECOMMENDATIONS OF THE INTERNATIONAL COMMISSION ON RADIOLOGIC PROTECTION. Brit. J. Radiol. Suppl. 6 (1955).
2. SIEVERT, ROLF M.: Medical Radiophysics in Sweden 1920—1950. Acta radiol. 33 (1950), 191.
3. SIEVERT, R. M. and WALSTAM, R.: Eine Methode für umfassende laufende Strahlenschutzmessungen. Fortschr. Röntgenstr. 75 (1951), 168.
4. SMITH, S. W. and KENNEDY, R. J.: Dose to walls in radiographic rooms. Radiology 64 (1955), 114.

BOOK REVIEWS

DIAGNOSIS OF CONGENITAL HEART DISEASE. By S. R. Kjellberg, E. Mannheimer, U. Rudhe, and B. Jonsson. 649 Pp. 581 Illust. The Year Book Publishers Inc., Chicago 1955. $ 22. —.

This is a text-book and as such is indispensable for all and sundry interested in the field of congenital heart disease. But it is more than this. In many respects it is primarily a scientific work containing many new and important observations.

The authors, two cardiologists and two roentgenologists, form a team which has investigated 396 cases of congenital heart disease. The authors give "an account of the results obtained with the diagnostic methods used, with particular emphasis on the special technical aids which make possible a detailed functional and anatomic diagnosis. Evaluation of the findings at the customary clinical examinations has been greatly facilitated by this means." MANNHEIMER and JONSSON are responsible for the clinical investigations including phonocardiography and cardiac catheterization. KJELLBERG and RUDHE have carried out the roentgenologic studies while the latter has analyzed the electrokymographic findings.

It is quite impossible in a short review to deal anything like adequately with the interesting matter in this comprehensive book even if one confined one's attention to the roentgenologic portions.

Two general chapters on embryology and the roentgenologic anatomy and physiology of the heart are followed by a description of the technique employed by the authors. The findings at angiocardiography form the foundation of the work. The technique used by the authors is based upon two principles, one of which consists in the injection of the opaque medium into the area involved or as close to it as possible *i. e.* selective angiocardiography, and the other in employing very rapid series of exposures. With this technique, the investigations have become so precise as to be compared with anatomic dissections. In many cases they may even surpass the latter, giving as they do information on live conditions.

Cardiac catheterization has been performed in the main according to the accepted technique. Those parts of the book dealing with electrokymography consist to a large extent of pioneer work and contain new and important observations. This method of examination as a matter of fact has not been employed previously in congenital heart disease other than in exceptional cases. The authors with due regard to the angiocardiographic findings, analyze the possibilities of visualizing the contours of the individual heart chambers and in doing so level considerable criticism at those optimistic authors of text-books who claim to be able to recognize every detail without much difficulty.

The authors, like all others performing catheterization and angiocardiography, have encountered complications. They maintain, however, that the risks are very small if the patients are in good condition: the more seriously ill they are, the greater the risk. It is doubtful whether the investigation was the cause of death in the grave cases; thus, two patients of the authors died before the examination on the day on which catheterization had been planned to take place.

The material is divided up into 24 different diagnoses with pulmonary stenosis, tetralogy of Fallot, atrial and ventricular septal defects, patent ductus arteriosus, and coarctation of the aorta, forming the largest groups. Every congenital defect is dealt with in a chapter by itself under the following scheme: pathologic anatomy and physiology, clinical symptomatology including phonocardiography and electrocardiography, conventional roentgenography, electrokymography, cardiac catheterization and angiocardiography. Every section contains a wealth of facts and expressions of opinions.

The book is profusely illustrated. The number of figures reaches 581 but as each consists on an average of three reproductions one can reckon on an actual total of more than 1,500 illustrations, an imposing figure and one which affords some indication of the

abundance of observations upon which the book is based. The illustrations are of varying quality. The photographs and reproductions of the anatomic specimens are excellent and the same applies to the electrokymograms. The reproductions of the conventional roentgenograms are poor and do little credit to the originals. All too often it is difficult or impossible to pick out details which are given in the legends. The angiocardiograms also fail to appear to advantage in the reproductions in the book. The originals are, however, of such superb quality that no poor reproduction or printing can obscure their character of perfected roentgenologic technique. This collection of films is a convincing demonstration of the superiority of the selective technique over other methods of angiocardiography.

Gunnar Jönsson.

Diego Carlström: X-Ray Crystallographic Studies on Apatites and Calcified Structures. 59 pages, 5 tables and 2 plates. Acta radiol. Suppl. 121, Stockholm 1955. Sw. Kr. 25: —.

Several calcium phosphates having an apatite structure have been proposed by different workers as the main constituent of the bone salt. In the present investigation the structure and other properties of these apatites are discussed, and it seems that there is only one, hydroxyapatite-$Ca_{10}(PO_4)_6(OH)_2$, which has well-defined crystallographic and chemical features. Several authors have suggested that the carbonate in some mineral apatites (carbonate-apatites), as well as in the bone salt, substitutes for PO_4-groups in the lattice. However, it was found that the carbonate in mineral apatites might occur as an almost amorphous calcium carbonate and in the case of bone and tooth enamel it seemed to be bound as CO_3-groups on the surface of the microcrystalline calcium phosphate.

The size of the apatite crystallites in bone tissue was determined using two independent roentgen diffraction methods: (1) line-widths measurements in the high-angle region using a Geiger-Müller diffractometer and (2) measurements on the diffuse particle scatter in the low-angle region. With the first method only the length of the crystallites could be determined, but the value found (230 ± 20 Å) was in good agreement with that calculated from the low-angle particle scatter (220 Å). This latter method also allowed an estimation of the diameter of the probably rod-shaped crystallites of bone salt (65 Å). The very small size of the calcium salt crystallites in bone leads to an enormous surface area (about 130 m^2/g bone salt) and a great number of different ions and water can be bound to the surface of the apatite crystallites thus contributing to the over-all composition of the inorganic fraction of the osseous tissues. There is certainly a close relationship between the inorganic crystals and the collagen fibres in bone, and it is proposed that the 640—660 Å repeating unit in the collagen fibre structure includes three apatite crystals having their long dimension (about 220 Å) parallel to the direction of the fibres.

In addition to the investigation on bone tissue, the composition of other calcifications such as osteogenic sarcoma and calcium salt deposits in blood vessel walls were analysed with the roentgen powder diffraction technique. In all these calcifications the calcium salt was shown to be in the form of microcrystalline apatite, indistinguishable from that in normal bone. Statoconia, the small crystalline bodies serving the static organ of sense in the inner ear, were found to be composed of single crystals of calcium carbonate. In mammals and birds these were in the form of calcite, whereas amphibians and bone fishes had statoconia composed of aragonite. In some very primitive fishes (lampreys) the statoconia consisted of calcium phosphate in the form of apatite.

Autoreview.